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## THE CONTROL OF INFECTIOUS DISEASE, WITH SPECIAL REFERENCE TO THE ROLE OF THE HOSPITAL.[1]


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INFECTIOUS DISEASES still comprise an important section of medicine. General practitioners to whom I have spoken estimate that 75% of the medical illnesses with which they have to deal are infective in origin. I congratulate the organizers of the Congress on their wisdom in including this plenary session in the programme. To illustrate the importance of the subject I will quote two recent statements. The first is from an editorial in the *British Medical Journal* commenting on an interim report of an inquiry initiated by the late Sir James Spence, the object of which was to keep under review the health and sickness experience of 1000 families in Newcastle (United Kingdom). The interim report deals mainly with the first year of life. I quote:

> . . . the predominant impression left upon the reader is the immense and continuing significance of infectious disease; 96% of all illnesses were infective in origin.

Then from the United States of America the *Statistical Bulletin of The Metropolitan Life Insurance Company* states:

> Although spectacular progress has been made in the control of infectious diseases, they are still responsible for an appreciable loss of life among children. Pneumonia and influenza and acute poliomyelitis are among the leading killers at ages 5 to 14 but the death toll from these diseases varies from year to year with their prevalence and virulence. Tuberculosis and the principal communicable diseases of children, while continuing their long-term downward trend are still far from negligible causes of death amongst youngsters. Moreover, most of the deaths from heart disease among this age range are of infectious origin.

Many of us feel that the position as regards acute infective medical conditions in the community is not entirely satisfactory.

An unexpected result of the success of antibiotic treatment against acute infections has been that more and more of the management of these diseases has been placed in the hands of general practitioners. While I agree that in many cases the general practitioner has encouraged this practice and carries out the treatment quite successfully, I wonder perhaps whether this is altogether in the best interests of the community in general and for the control of communicable diseases. One of the functions of the old-fashioned fever hospital was the isolation of the patient. I agree that as a method of control of epidemics isolation was of perhaps minor importance. On the other

[1] Read at a plenary session on "Control of Infectious Diseases", Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August, 1955.

hand, the present method of attempting to manage all acute infections in the home has almost as many dangers. The risks of intrafamily spread should always be considered.

### Streptococcal Infections.

For example, a tendency has developed to treat patients with streptococcal infections rather lightly. I agree that they respond well to penicillin and, if treated promptly, come to no immediate danger. I am not sure, however, if it is sufficiently realized that the streptococcus is still an infection which, if not recognized and treated immediately, may produce acute nephritis or acute rheumatism. Do we realize the risks to the rest of the family when a child with a streptococcal infection is given a few shots of penicillin and allowed to mix freely with other children?

Let me quote two examples of outbreaks in which the first indication of streptococcal infection in a localized community was the occurrence of multiple cases of nephritis. The work of Rammelkamff and Dingle, which has been verified by the laboratory at Fairfield, has shown that there are at least two types of streptococci which can be nephritogenic.

Similar dangers exist in regard to rheumatic fever. It is a dangerous practice to treat an acute streptococcal infection as of no consequence. Penicillin should be exhibited as soon as possible; and if the patient is in a crowded household, it is wiser to move the patient to hospital before others develop the infection. The same risks exist in a general hospital, where the patient should be isolated with barrier nursing precautions. The results of spread of a nephritogenic type of streptococcus in a hospital ward could be catastrophic.

### Infectious Hepatitis.

Another example is infectious hepatitis. I doubt if it is sufficiently appreciated what a disaster the introduction of infectious jaundice may be to a family. If a child develops infectious jaundice in a household, serious consideration should be given to administering $\gamma$ globulin to the adults and to sending the patient to hospital unless he can be safely isolated for fourteen days. Adults develop jaundice more severely than children and are more prone to develop recurrences. Convalescence is often long and irksome, and the economic loss is serious. The ultimate prognosis is often doubtful. The management of a severe case of jaundice in an adult is better carried out in hospital, where biochemical tests may be used as significant checks on clinical findings. Jaundice is now a first-class public health problem. The family group is usually infected from the school; and when the infection is introduced into a household, the attack rate in the home is much higher than in the school.

### Other Infections.

The general practitioner is now called on to treat and investigate all kinds of acute infections, often without adequate laboratory assistance, and usually has not the time to investigate the epidemiological cause of the infection.

It is my contention that he needs help: (*a*) by better laboratory assistance, (*b*) by increased hospital facilities, (c) by a closer liaison with the health department. I feel that all these needs can be best supplied by a specialized hospital. Nowadays there is a tendency to belittle the need for specialized infectious diseases hospitals. From the experience of the last few years and from the number and variety of acute infections which practitioners and hospitals now send in to Fairfield, there seems to be greater need than ever for such a hospital. Used properly it is the key point between the general practitioner and the health department. Samples of all acute infections occurring in the community may be there subjected to expert clinical, laboratory and epidemiological investigation.

In Melbourne many general practitioners are becoming increasingly aware of the advantage of the facilities at Fairfield for admission and investigation of their patients. A close liaison by consultation and letters is being established between the general practitioner on the one hand and the preventive aspect of the health department on the other. It is, of course, essential that the hospital laboratory must be of top quality staffed by experts who are clinically minded.

The advantages of a specialized hospital are: (i) accurate diagnosis, (ii) specialized management of severe infection, (iii) centralization of cases for epidemiological study, (iv) assessment of results of preventive inoculation, (v) scientific evaluation of results of specific treatment, (vi) publication of reports and presentation of patients at clinical meetings, (vii) isolation of patients as a means of diminishing intrafamily spread, (viii) provision of a headquarters of clinical and laboratory research.

Some recent observations may be of interest.

### *Whooping Cough.*

In a severe recent epidemic of whooping cough an estimate of the value of prophylactic vaccination against whooping cough has shown that it is of definite value, but that there is still room for improvement in the quality of the vaccine. Many of us would welcome further trials of the vaccine containing pertussis hæmagglutinin, work on which was initiated by Keogh, North and Fisher in Australia.

### *Non-Paralytic Poliomyelitis.*

In Melbourne in late 1952 an outbreak of mild headache, neck stiffness and vomiting occurred in many suburbs. The illness lasted only a matter of two to three days, and no signs of paralysis developed. A practitioner in an industrial suburb told me that he and his partners had met with no less than 60 of these cases in a week-end. A number of the patients were admitted to hospital, and lumbar puncture showed that most had increased cells in the cerebro-spinal fluid. Subsequent examination in the tissue culture laboratory by Dr. Ferris showed a high proportion of isolations of Type 2 poliomyelitis virus. Epidemics of Type 2 are extremely rare, and as was shown in this epidemic the proportion of severely paralysed patients is low. The amount of natural immunization against Type 2 must have been very extensive. This epidemic has been described for the journal by Dr. John Forbes.

### *Bronchopneumonia.*

At present in Melbourne a wave of cases of bronchopneumonia is sweeping through the city. At the same time a measles epidemic is in progress. The percentage of cases of measles complicated by pneumonia is high. For some weeks prior to this, comments had been made on the frequency of throat swabbing from patients which contained significant numbers of pneumococci. This increase indicated an unusual carrier rate in the community, and it is reasonable to accept this as the cause of the outbreak. The early exhibition of penicillin in young children and elderly adults is the obvious public health answer to the problem.

### *Gastroenteritis.*

Considerable increase in non-seasonal bowel infections has been noted in Melbourne, occurring both in children and in adults. The increase is mainly in salmonella infections of various types, but most commonly those due to *S. typhimurium*. These infections respond irregularly to "Chloromycetin", and the infection once present in the bowel appears to remain indefinitely. The accumulation of carriers in the community must be reaching fearsome proportions.

### Country Hospitals.

Before concluding, I should mention the country hospital with an isolation wing which often because of lack of staff is not being used. I would emphasize the risk in nursing patients with such conditions as infectious hepatitis, acute respiratory infections and gastro-intestinal infections in general wards. The incubation period of infectious hepatitis is about thirty days. The contacts of a patient with jaundice will be usually discharged from hospital before the end of the incubation period. The

hospital authorities may thus remain blissfully ignorant of any infection occurring as a result of cross infection. Similar risks are sometimes taken with acute respiratory infections.

Professor Hare has entitled one of his lectures given in Australia "The Hospital as a Reservoir of Infection". I am afraid that in some respects the inference may be true. Yet there is no reason why cross infection of medical conditions should occur in a hospital in which simple isolation principles can be practised. A system of graded isolation, such as that in use at Fairfield, simplifies the procedure and cuts down the time wasted in barrier nursing.

To summarize, I have tried to make it clear that the specialized fever hospital plays an even more important role than formerly in the treatment and control of acute medical infections in the community.

## CONTROL OF INFECTIOUS DISEASES.[1]

By D. W. JOHNSON,

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BEFORE presenting to you some ideas on control of infectious diseases from the viewpoint of a public health administrator, I want to get back to first principles and define an infectious disease. Strictly speaking, an infection is invasion of tissues by pathogenic organisms. An infectious disease is one whose signs and symptoms are produced in the host by invasion of a parasitic agent. If the disease is capable of being communicated by infection to others, it is a communicable disease. Chickenpox is a communicable disease, tetanus is not; yet, as we shall see later, chickenpox is not notifiable, and tetanus is notifiable.

What, then, is a notifiable disease, and why are not all communicable diseases notifiable? A notifiable disease is a disease which a Health Department states shall be notifiable in writing. Most of these are communicable diseases, but others (such as lead poisoning, hydatid disease, trichinosis and tetanus) are not. What is the purpose, then, of notification? To answer that intelligently, let us consider the fact that 46 diseases are notifiable throughout Australia. Of these diseases, 40 are notifiable in South Australia, while only 24 are notifiable in New South Wales. The position in the other States falls between these two extremes.

Seventeen diseases are notifiable in all six States, and these include the exotic epidemic diseases (such as smallpox, cholera and plague), whose periodic visitations in the nineteenth century caused great public alarm. In addition, diseases which are or have been endemic, and sometimes epidemic (such as diphtheria, typhoid and tuberculosis), are included in this group.

Nine diseases are notifiable in five States. New South Wales does not consider notifiable such diseases as anthrax, malaria, amœbiasis and bacillary dysentery, though these diseases are certainly present, whilst Western Australia does not require notification of puerperal fever.

Seven diseases are notifiable in four of the six States. Rather surprisingly, dengue fever is not notifiable in Queensland. (This omission is receiving attention.) Tetanus, an eminently preventible disease, is not notifiable in New South Wales or Tasmania. At the bottom of the list are those diseases which are notifiable in from one to three States. It is interesting to note that pertussis and epidemic influenza are notifiable only in Victoria. Clearly, there is no uniformity amongst the States on what diseases should be notifiable.

[1] Read at a plenary session on "Control of Infectious Diseases", Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August, 1955.

The original purpose of notification, still valid today, was to enable some central or local health authority to become aware that cases of communicable disease were occurring in a particular locality. The public could then be warned, and, if possible, measures could be taken to prevent spread of infection.

The control of spread of infectious disease was the reason why most of our State Health Departments came into existence. With some of these diseases control measures are effective to a varying degree, but how can lead poisoning or tetanus be controlled if they are not communicable? The answer is that they can be entirely eradicated. So can diphtheria and malaria and some other diseases. However, this idea of notification to secure the eradication of disease, and to check on the completeness of eradication, is relatively new; for, except for smallpox, there was no method for eradicating disease until about thirty years ago.

### Smallpox.

Smallpox visited Australia periodically from about fifteen months after the arrival of the First Fleet in 1788 (Tidswell). The disease spread to the aborigines, and it is estimated that at least half the aboriginal population in south-eastern Australia had succumbed to the disease by 1850 (Cumpston, 1914). Every State except New South Wales introduced legislation providing for compulsory vaccination. The last major outbreak occurred in 1881 and 1882, though occasional cases were reported from all States until 1909. Since then, no case has been reported in which the patient contracted the disease on this continent.

Freedom from smallpox has rendered most of us complacent, yet I am not at all sure that this attitude is justified. It is true that no subclinical cases of the disease are known to occur, but alastrim or even smallpox itself in a vaccinated subject can be very difficult to diagnose. It is entirely possible for the virus to enter this country and not be observed by quarantine inspection. It has been introduced in this way into Great Britain.

The subject of compulsory vaccination has been raised by various authorities from time to time. The National Health and Medical Research Council examined this question in 1946 and again in 1955, and on each occasion they recommended against universal vaccination, holding that the risk of serious complications following vaccination was rather greater than the risk of the introduction of smallpox virus. Instead it was recommended that persons who would be most likely to be contacts of an introduced case of the disease (doctors, nurses, customs officers, transport personnel and police) should be offered free vaccination. Probably this is all that is required. Yet, as an epidemiologist, I can only contemplate the present position—that of an almost totally unprotected population—with considerable disquiet. Speaking personally, I should like to see more babies vaccinated in this country.

In the first year of life, complications of vaccination are extremely uncommon.

I know that a million doses of smallpox vaccine are kept in Melbourne to cope with an outbreak of smallpox in this country, yet I would be much happier to know that some of it was being used to vaccinate even 25% of the 200,000 babies born in Australia each year. I commend smallpox vaccination of infants to the practising profession, and I hope that some of you will advocate vaccination when parents ask for advice.

Reviewing the history of smallpox in Australia, Cumpston (1914) states:

> There have been other times when no quarantine organization, however perfect, could have prevented the introduction of the disease. That such occasions as these latter will be many times repeated in the future, is in the nature of things inevitable.

### Plague, Cholera, Yellow Fever.

I will pass over plague, cholera and yellow fever because the risk of their introduction to Australia is remote under present quarantine regulations.

### Diphtheria.

Throughout Australia, the incidence of diphtheria has reached a very low level. Too many people are inclined to think that active immunization is the cause of this happy position. How true is this? Victoria has recorded deaths from diphtheria and croup since 1853. Until 1858 diphtheria was not diagnosed. In that year diphtheria became epidemic in London, though in 1861 Jenner stated that he had seen sporadic cases "ever since he practised medicine, and the writings of the older English physicians prove that from time to time it has been epidemic, or very common, in many parts of England".

At all events, a severe outbreak of diphtheria commenced in Victoria in October, 1858, and thereafter caused heavy loss of life in children. The behaviour of the disease in the five years following 1858 resembled that of a virulent disease in a susceptible population. In 1860 it caused 792 deaths. From then on, even though heavily endemic in intervening years, diphtheria epidemics occurred every three to seven years, but never with the severity of the original outbreak. In fact, the mortality showed a general downward trend even before the introduction of antitoxin, first supplies of which arrived in 1895.

In Queensland the same trend was evident. In spite of increase in population, deaths in Queensland from diphtheria and croup had declined from 1031 in 1890 to 94 in 1923, so that the death rate was already at a low level before the introduction of active immunizing agents. Without immunization, diphtheria probably would have continued to decline, although not at the same rate, until another pandemic of diphtheria occurred and spread to Australia. It is still not generally realized that the introduction of active immunization coincided with a decline in incidence of diphtheria. The natural history of diphtheria is one of fluctuations and cycles, and it is unrealistic to believe that it is banished forever.

Diphtheria is declining for two reasons. The first reason is that *Corynbacterium diphtheriæ* is recovered from fewer and fewer throats in routine testing. This experience is world wide, and in its way is quite exciting news. It means that carriers are fewer, and so opportunities for the disease to spread are severely curtailed. The second reason is that active immunization prevents the development of clinical disease in the great majority of immunized children. This is true only if basic immunization in infancy is followed by reinforcing (or "booster") doses of prophylactic material at appropriate intervals. Probably reinforcing doses should be given at about five and ten years of age; but if diphtheria again became prevalent, I would not hesitate to advocate more frequent doses of toxoid. I would also advocate that pregnant women who had been immunized in childhood should be given a reinforcing dose of toxoid, so that the babies in these bottle-fed days would start life with some circulating antibodies against diphtheria. In the face of a severe outbreak, I would favour active immunization of adults.

The incidence of diphtheria is now so low that it is easy to believe that the population is protected. The immunity conferred by active immunization wanes, and little natural infection is occurring to maintain immunity. Ipsen and Bowen (1955) have shown that about 50% of the adult population studied in Massachusetts are susceptible, and I have no doubt that a similar finding could be made in Australia. The reservoir in children is being eradicated, and more adults are becoming susceptible. Diphtheria could therefore occur again in epidemic form, but in diphtheria toxoid, properly used, we have a weapon to control it.

### Whooping-Cough.

Prevention of whooping-cough is imperative in the first year of life, and therefore pertussis vaccine should be given at two to three months of age. If pertussis was prevalent, I would give vaccine to a child one month of age.

The value of vaccination against whooping-cough is now well established. Most immunized children will not acquire the disease if exposed to infection, and most of those who do develop whooping-cough will have a mild infection. Furthermore, an immunized child can be given a "booster" dose of vaccine if known to be exposed to infection.

It is true that pertussis vaccine sometimes has undesirable side reactions. For instance, quite severe local and constitutional effects can follow a dose of vaccine. Even more important is the occasional development of encephalopathy. These disadvantages are not sufficiently numerous or serious to justify withholding routine administration of the vaccine.

### Tetanus.

Tetanus is relatively uncommon, but each year between 30 and 50 cases of the disease are notified in Queensland. Natural infection leaves no titratable antibodies. Queensland, with less than 15% of the population of Australia, contributes almost 30% of the deaths from tetanus in Australia, and deaths from tetanus exceed deaths from diphtheria and whooping-cough combined. About 50% of cases are in children under the age of fifteen years, and the overall mortality rate exceeds 50%.

Tetanus is a completely preventible disease, and for several years I have been an enthusiastic supporter of active immunization. I have heard objections that the risk of contracting tetanus is not worth the cost of mass immunization of children. This is a superficial and materialistic view, and, moreover, is no longer valid when tetanus toxoid is combined with other immunizing agents as in triple antigen. Every child should be immunized against tetanus, and antibodies should be kept at a high level by booster doses at appropriate intervals.

What is an appropriate interval? The American Army recommends every four years. The American Public Health Association recommends an interval not longer than five years. Recent work by Peterson *et alii* (1955) showed that only 30% of a group immunized more than six years previously had serum antitoxin levels below 0·1 unit, which is considered the protective level. In the absence of injury, four or five years should be considered the appropriate interval between "booster" doses.

In addition to the feeling of security given to parents, proper immunization against tetanus affords much relief to the doctor called upon to treat a wound in which infection with tetanus spores is possible. Routine treatment of such a wound in an unimmunized patient includes administration of tetanus antiserum, and each administration carries with it the risk of serum anaphylaxis. A severe reaction not infrequently results in death, and the doctor gets little credit for this. Severe sensitivity reactions are likely to develop in patients who have had previous experience with horse serum or who have personal or family histories of allergy. Laurent and Parish (1952) have described very clearly the various types of reaction to serum, and the procedures they recommend to avoid these should be widely adopted.

If, on the other hand, the patient has received basic immunization against tetanus, there should be little doubt about what procedure the doctor should adopt. It is clearly established that the level of antibodies rises significantly within five to six days after a stimulating dose of tetanus toxoid, and this is a shorter period than the average incubation period of tetanus. If the doctor is satisfied that the wound is less than twenty-four hours old, that it carries a risk of tetanus, that the patient has received satisfactory basic immunization, and that he has not received a booster dose of tetanus toxoid within four or five years, he should give a stimulating dose of toxoid and feel confident that tetanus will not develop. Only if the wound is more than twenty-four hours old, or if the wound has caused great tissue destruction, should he give tetanus antitoxin (minimum dose, 10,000 units) in addition. This procedure is now standard in the British and United States armed forces.

### Intestinal Bacterial Diseases.

Intestinal bacterial diseases include typhoid fever, paratyphoid fever, infections with Shigella and Salmonella organisms, and infections with the varied organisms that seem to be responsible for infantile diarrhœa.

Typhoid fever, which caused the deaths of between 1000 and 1600 persons annually in Australia during the last six years of the nineteenth century, and which filled the typhoid wards of our hospitals, caused seven deaths in 1953. Yet the population as a whole is as susceptible now as then, and active immunization against typhoid is not widely practised outside the armed services. Is the decline due entirely to improved methods of control? I am inclined to doubt it. Probably, like diphtheria, the organisms causing typhoid have become much less frequent as a result of a natural fluctuation of the disease. When typhoid was common, there were many carriers. Now there are few. On the other hand, it is idle to deny that this century has witnessed a dramatic improvement in standards of living and of hygiene. Food, milk and water are all much safer than they used to be. But if standards of living and of hygiene were responsible for the decline in typhoid, why have not diseases due to other intestinal pathogens decreased? Instead, there is considerable evidence that infections with Salmonella and Shigella organisms not only are not declining, but are increasing.

It appears that organisms wax and wane in communities. For example, as infections due to *Shigella sonnei* decline, infections with Salmonella organisms take their place, and the large number of symptomless or convalescent carriers renders control difficult except in closed institutions. It is essential to break the chain of infection from anus to mouth, and various measures will achieve this.

The most important lessons in elementary hygiene are often the ones that are least observed. Take the case of a person with an acute respiratory infection: he knows very well that he should not be in contact with the public, and that his nasal discharges are infectious, yet he rarely stays at home with a cold. Likewise, a person who defæcates generally considers he has done his duty to himself and to others when he uses toilet paper. I believe it is true that the average Australian does not wash his hands after leaving the toilet. It will take a great deal of propaganda to make this a routine performance. We may try to instil it into school children; but is the average public school lavatory equipped with hand-washing facilities? For that matter I believe that the majority of public sanitary conveniences are not equipped with towels and wash-hand basins.

The people who prepare and handle food in restaurants and hotels are often not careful in toilet hygiene. Until the washing of hands after the toilet and before handling food becomes a national custom, we can expect a high incidence of intestinal diseases of bacterial origin.

### Scarlet Fever.

Pickles (1939) states:

> As is well known, scarlet fever has passed through many fluctuations in severity. I happen to possess John Pechey's edition of The Whole Works of that Excellent Physician, Dr. Thomas Sydenham, printed in 1701, and it is interesting to see scarlet fever referred to as "this Name of a disease, for it is scarce anything more", and many of us in this period would hold the same opinion of the disease as we have seen it.

He goes on to discuss scarlet fever in the nineteenth century and comments on "the very real fear which is still felt in our district where memories are long and the tragedies of the past are never forgotten. Scarlet fever in those days was 'fever', i.e. the fever, and required no qualifying adjective any more than, as Greenwood writes, the plague requires one".

These fluctuations in virulence have also occurred in Australia. Deaths from scarlet fever in Victoria increased from an average of 32 for the five years ending in 1859 to 461 in the next five years, and thereafter remained at a high level until 1870. A severe epidemic in 1876 caused 2240 deaths, the death rate reaching the high figure of 280 per 100,000 population. Thereafter it declined, and apparently the organism assumed its present mild form.

In the whole of Australia during 1953 there were only three deaths from scarlet fever. It is quite apparent that a marked decline in the virulence of the infecting organism has occurred, just as happened in the eighteenth century in England. How long will the present mild phase last? I doubt if anyone can give an accurate answer.

While scarlet fever is in its present mild phase, control is relatively unimportant. In fact, some doctors say: "Why bother to notify scarlet fever at all?" It is true that because a child is unfortunate enough to be sensitive to erythrogenic toxin he develops a rash, while his brother or sister with the same organisms in the throat develops only a sore throat. Therefore only a small proportion of streptococcal infections are notified. Those of us who remember the past history of scarlet fever agree that notification of scarlet fever, even if incomplete, is the only index of the prevalence of streptococcal infections. While the disease is notifiable, change in virulence can be noted quickly, for I am convinced that a virulent phase will recur.

It has been now established that penicillin is effective in controlling spread of the disease in closed institutions. I would certainly not hesitate to use it in order to protect a particular contact in whom infection with the scarlet fever streptococcus might be particularly undesirable, or in order to end an epidemic in a military camp or boarding school. I am not so sure that oral or depot penicillin therapy is advisable merely to protect school or family contacts. After all, it is impossible to prevent streptococcal infections at some time during life. It is probably better to let a child be exposed to the present mild strains, so that he has an opportunity to develop immunity at an age when the incidence of complications of the clinical disease is insignificant.

### Tuberculosis.

Deaths from tuberculosis have fallen dramatically in the last fifty years. For instance, in Queensland, whereas in 1900 tuberculosis was responsible for more than 9% of all deaths, in 1954 the figure was little more than 1%. Other States can show a similar decline.

Modern methods of treatment with chemotherapy and surgery were not used until about ten years ago, so therapy cannot be responsible. Although contact case finding techniques have been used over a longer period, they were not intensively applied until the post-war years, and it is questionable whether they have been a significant factor in the decline of tuberculosis since the turn of the century. There is no decline in the virulence of the tubercle bacillus. Tuberculosis in susceptible native races is still a fairly acute disease whenever recently introduced. Most authorities believe that the important factors are nutritional and genetic. The average Australian today eats a wider range of foods, works under better conditions, and has better housing than did his grandparents. The genetic factor, however, is more important. Exposure of successive generations to the tubercle bacillus has tended to weed out the susceptible fraction of the population.

The excellent nation-wide tuberculosis programme will ensure that the present decline in tuberculosis will be accelerated until tuberculosis will become relatively uncommon by the end of this century. What problems will then require to be faced? The principal one is that a majority of the population will go through life without developing a positive reaction to the Mantoux test. Are they then to be regarded as susceptible to tuberculosis should they be exposed to the tubercle bacillus? In other words, will it be possible for tuberculosis to assume epidemic form again? I think not. There is no evidence that the susceptible minority will outbreed the resistant majority, and it appears likely that tuberculosis, like leprosy today, will become a rare disease in Australia. Before this stage is reached, a better immunizing agent than B.C.G. vaccine may have been developed.

### Poliomyelitis.

Poliomyelitis has been a headline item for several years, and in April of this year the publication of the Francis report on the Salk vaccine took up more newspaper space than did the announcement of the atom bomb in 1945. Yet, as a killer, poliomyelitis ranks low among the causes of death.

What are the basic facts about poliomyelitis? The disease is about as infectious as chicken pox, and spreads rapidly in contacts, particularly in the home. A few days after the introduction of virus into a household, most members of the family will have the virus in the pharynx or in the alimentary tract, but it is rare for more than one member to develop clinical disease. In primitive communities, infection with poliomyelitis virus occurs early in life, and the usual outcome is immunity through subclinical infection. Thereafter throughout life, the virus probably repeatedly parasitizes the intestinal tracts of the community, and a high level of immunity is maintained. In Western countries, the better standard of hygiene prevents contact of the virus with most children until they start school, because the peak years of infection are the first five years at school. Many children appear to escape infection altogether with the virus, and develop clinical disease in adult life.

Hence, in Australia, there is this constant change in age pattern of the disease to the older age groups. At present about 35% of patients with poliomyelitis are over the age of fifteen years, and I feel that this "shift to the right" will continue.

In considering the efficacy of quarantine measures, it is important to keep in mind the probability that every household contact is infected by the time the patient develops signs of central nervous system involvement, and that the average duration of virus excretion will be several weeks. It is now practically uniform procedure in the various States to enforce house isolation of house contacts under the age of sixteen years for a period of two weeks from last contact with the patient. It is also recommended that contacts over the age of sixteen years who handle food (for example, waitresses, milk industry employees) should cease work for fourteen days. The World Health Organization (1955) in its monograph on poliomyelitis recommends that quarantine of contacts be maintained for twenty-one days. The Ministry of Health in Great Britain advocates this period. Perhaps Australian States will extend the quarantine period to three weeks.

To me it seems that isolation of contacts of poliomyelitis is a reasonable procedure. It is true that not all carriers or excretors of virus will be controlled by this measure, but at least isolation will prevent dissemination of virus by contacts who are almost certainly infected. In this way the march of an epidemic through a community might be appreciably slowed.

I will say little about poliomyelitis vaccine. I believe that active immunization against poliomyelitis will be achieved ultimately by using living attenuated or modified strains of virus, and vaccines of this type are in course of development in America. In the meantime, the Salk vaccine, properly prepared and incorporating the improved techniques developed since the 1954 trials, is at least as effective as whooping-cough vaccine and will probably confer immunity of longer duration. When supplies are available, it should be given to every child.

What will be the position when every child is successfully immunized against poliomyelitis? Paralytic poliomyelitis in children will become as rare as clinical diphtheria in children is today, yet there will be an increasing number of susceptibles amongst the adult population. Will the adult population be sufficiently protected by control of the disease in children, or will we witness outbreaks of poliomyelitis confined to adults? The position, as I see it, will be that like that of diphtheria; and if a virulent strain of poliomyelitis is introduced into a community with a large percentage of susceptible adults, the widespread use of poliomyelitis vaccine in adults must receive very serious consideration. However, our immediate objective should be the active immunization of children.

### Other Communicable Diseases.

I have not attempted to deal with the control of all communicable diseases. I have omitted the acute respiratory infections, not because they are unimportant, but because efficient methods of control have not yet been devised.

I must mention briefly the prevention of rheumatic fever. This has assumed new importance now that it is clear that neither cortisone nor corticotropin is curative. The risk of recurrence in a patient who has had one attack of rheumatic fever is estimated as 60% to 75% in the three years following the initial attack. The risk appears to be directly associated with respiratory infections (either mild or latent) due to group A streptococci. There is now evidence that the risk of recurrence is greatly reduced if group A streptococci can be eliminated from the upper part of the respiratory tract, and this can be achieved by using sulphonamide drugs or oral or depot penicillin therapy. Treatment must be continued for several years, and there is little doubt that the recurrences of this crippling disease will become less common as more and more patients are put on prophylactic treatment.

### Discussion.

From listening to me, you may have gained the impression that I am a lukewarm supporter of some traditional control measures. That is only partly true. For instance, I do not advocate aerial disinfection in the home—not because disinfection is bad in principle, but because efficient disinfection cannot be performed without elaborate equipment. Therefore, it is better to admit this rather than to engender a false sense of security. I do not believe in isolating contacts of the minor diseases which cannot be controlled—for instance, measles, chicken pox, rubella and perhaps scarlet fever. With these diseases I feel that it is better that contacts and others should be allowed to develop them at an age when they are of relatively minor severity, rather than deliberately to postpone infection to an age when the disease may be severe and complications not uncommon.

When a disease is serious and when isolation measures are known to be reasonably effective, I do not hesitate to recommend isolation. Summed up, my creed is that active immunization should be universal for those diseases for which effective antigens have been developed. If active immunization is not available for a common mild disease, the sensible view is that a person should develop the disease at an age when mortality is least likely to occur. If a communicable disease is a serious one, such as poliomyelitis, quarantine measures should be utilized until the patient and his family contacts are probably not infectious to others.

In the prevention of communicable diseases it is very important that the public should be well informed, because intelligent public cooperation in control measures will ensure success. To secure this, health education is essential. It is no longer possible to scare people into doing something for fear of destruction from pestilence. In the past, the individual and the community submitted to harsh and often unproductive sanitary regulations. Today, the public want to know what the Health Department and the medical profession are doing, and they will cooperate, not from fear, but from their desire for a fuller and healthier life. Keeping the public informed is today an important function of a Health Department.

Progress may be gauged better by looking back than by looking ahead. If we look back a half century or a century, as we have done today in certain diseases, it will be clear that Australia has made considerable progress in controlling communicable diseases. Diphtheria, whooping-cough and tetanus can be reduced to a point where they are no longer of public health importance. Scarlet fever is now in a mild phase of its natural history, but the prophylactic and therapeutic weapons to control the severe phase (should it recur) are on hand. Tuberculosis is yielding to intelligent control and to effective therapy. Typhoid is rare, and, if it recurs, can be both prevented and cured. The acute bacillary intestinal infections can be controlled by intelligent public cooperation in better handling and storage of food, and by hand-washing after the toilet. Measles, mumps and chickenpox are minor infections if contracted in childhood. Infective hepatitis and acute respiratory infections are still uncontrolled. It is a pleasing retrospect, and the future will be better than the present.

What remains to be done in the future? There are diseases (such as respiratory and certain other virus diseases) which must be intensively studied before they can be effectively treated, controlled or prevented. It may also be shown that a considerable amount of impairment of good health is due to subclinical attack by various pathogenic agents. Farm animals gain weight and utilize food better if fed antibiotic supplements, and these antibacterial supplements apparently suppress organisms that were interfering with the health of the animal. Man must live with pathogenic organisms, and further research may show that his health is being affected by his failure to adapt himself to them.

In concluding, may I stress the important role of the practising doctor in prevention of disease? The practising doctor sees the sick patient, he makes the diagnosis, he goes into the home, and he advises the other members of the household. He has a unique opportunity to observe spread and to urge prevention. The practitioner of preventive medicine receives notifications of disease; he can classify these, he can observe trends, he may be able to advise on prevention—but he rarely sees the patient. With active cooperation, the practising physician and the practitioner of public health can achieve dramatic results in the control of infectious disease. We both serve the people, and our chief responsibility is to them. If we continue to work together in the future, as we have done in the past, we will justify the faith that the public now has in our profession.

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## SOME ASPECTS OF THE CONTROL OF THE DISEASES OF ANIMALS TRANSMISSIBLE TO MAN.[1]


By J. A. R. MILES,

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WHEN the control of the diseases of animals transmissible to man is being considered, the first point which must be clearly understood is that the zoonoses, as these diseases are nowadays commonly called, are diseases primarily of animals, and that man only accidentally becomes affected. He is quite unnecessary for the normal cycle, and his infection is usually a dead end for the parasite, which rarely succeeds in passing from man to man. Therefore the aim, merely from the point of view of human health, need only be to stop man getting in a position in which he is likely to be involved in the cycle of these diseases; but, unfortunately, this is not always easy.

However, from time to time, these diseases do succeed in passing from man to man, and may even make several passages in man; and, as Burnet has pointed out, such an occurrence may well be the way in which a parasite becomes adapted to man, and a new human disease becomes established.

Further, in the case of some diseases of domestic animals, it is difficult for man to avoid opportunities for infection; and even when the disease is one normally infecting wild animals, either through his own movements or through those of the animals, man may become involved in the cycle, and an epidemic may result.

Because human infection with zoonoses is not a simple matter of man-to-man transmission, there are greater opportunities of control than with such a disease as influenza, and the application of such knowledge as we have of their natural history may greatly reduce the number of infections with certain of the diseases and give us warning of the risks of infection with others.

In this paper I shall try to illustrate some of the ways in which we can use knowledge we already have to combat certain zoonoses, due either to viruses or rickettsia, which are known to occur in Australia. These diseases can be divided into those which are normally distributed by an arthropod vector, and those which are spread either directly from the animal host or through fomites.

### Diseases Transmitted by Arthropods.

Firstly I shall consider the diseases ordinarily spread by arthropods.

The most widely spread of these diseases in Australia is endemic or murine typhus, which probably is present in all States. It is a disease caused by a rickettsia very similar to that of epidemic typhus, but differing in that instead of having a cycle from man to man through human lice, normally *Pediculus corporis*, its usual cycle is in rats, spread by fleas and possibly also by mites and rat lice, and it only occasionally infects man when he is bitten by the rat flea, *Xenopsylla cheopis*.

The disease is apparently endemic in the rats, and a few human infections occur each year. Sometimes a group of cases may occur together, as if they were the overflow of some epizootic among the rats, but the natural history of this disease has not been thoroughly studied in Australia, and we do not know a great deal about the rodent cycle. However, the disease appears ordinarily to be mild in rats, and not to be a cause of highly fatal epizootics, like those which presage human epidemics of plague. The control of the disease is the control of the rat population, and provided the rodents are not allowed to breed up to very large numbers, not many cases of endemic typhus will be seen.

By contrast with endemic typhus, tsutsugamushi, or scrub typhus, is essentially a disease of bush rodents, and is transmitted by a variety of trombiculid mites. The disease is transmitted through the egg to succeeding generations of mites, and this is necessary for the survival of the organism, since these mites ordinarily take only one blood meal during their life. This meal is taken in the six-legged larval stage. Man becomes infected through the bite of an infected larval mite.

The disease is, therefore, a disease either of untouched bush or, even more, of areas once brought into cultivation and then allowed to revert to scrub. It has been recorded only from tropical areas with quite a high rainfall. As far as I know, in Australia it has been proved to exist only in Queensland.

In such areas there may be large populations of rodents and of trombiculids, and if the mites are infected with the rickettsia, anyone who is rash enough to remain in the area for a substantial time has a very good chance of suffering from scrub typhus. Except in Japan, few cases were seen before World War II, but at that time large numbers of susceptible soldiers were introduced into endemic areas and serious outbreaks resulted. Ordinarily the areas which are very dangerous for scrub typhus are limited in extent, and the danger arises when people are moved into a new area. In war time it is not possible to survey such an area properly, but in normal times it should be possible for public health authorities to visit an area scheduled for close settlement, or for a holiday camp, and to survey it for the existence of a likely focus of scrub typhus. If there is a suitable area, rodents could be trapped and mites collected to determine whether, in fact, the disease was present; and if so, either the area could be so cleared as to eliminate the danger, or those planning a holiday camp could be advised to try elsewhere.

[1] Read at a plenary session on "Control of Infectious Diseases", Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August, 1955.

A vaccine for the prophylaxis of this disease was developed during the war years, but it was never properly tested, and we still do not know how effective it could be in the field. However, we do know that it was capable of protecting laboratory animals, and that it did not give 100% protection to workers in the laboratory making the vaccine.

As with scrub typhus, the existence of a form of tick typhus in North Queensland was first revealed during the last war. The recorded cases of this disease have not been numerous, although late reports have indicated that the disease exists quite far south in Queensland. Epidemiological evidence suggests that the tick *Ixodes holocyclus* is the main vector involved in this disease, and Fenner's serological survey on the Atherton Tableland revealed complement-fixing antibodies in the sera of eight of 111 wild mammals tested. They included specimens from five different species, one rodent and the rest marsupials.

Up to the present this disease has been relatively unimportant, but the movement of large numbers of men into a highly endemic area following a mineral find could lead to a large number of cases. A preliminary biological survey of the area, such as I have suggested in the case of scrub typhus, would give warning of the risks involved and a chance for the health authorities to take suitable precautionary measures to reduce the risk.

Murray Valley encephalitis is a disease of wild birds, and is almost certainly transmitted by *Culicine* mosquitoes (McLean has shown that all the *Culicine* species he tested in the laboratory were capable of transmitting the disease). The nature of the animal reservoir and the vector made this a disease capable of wide movements, and human epidemics have occurred when the disease has spread to areas with a substantial human population.

Anderson and Miles have independently developed essentially similar theories of the natural history of this disease. I quote my own conclusions with small modifications. The virus is a virus of wild birds, in which it normally causes a mild non-fatal disease, with either brief or fairly prolonged viræmia. It is almost certainly spread from bird to bird by *Culicine* mosquitoes. In seasons with exceptional early rains, when birds breed early and second broods are usual, early movements of young birds to the south occur; many of these birds may be infected, and when they arrive at suitable destinations in the season when mosquitoes and susceptible juvenile locally bred birds are numerous, they initiate an epizootic. When, either by direct flight or through a chain of infection arising from shorter dispersal flights, the birds initiate an epizootic in a more densely settled area, man and his domestic animals may be casually infected. The exact area in which the epidemic occurs will depend on the arrival of infected birds at a time optimal for spread of the disease.

More recently, Miles and Dane have found evidence from two areas in the Northern Territory that the virus of Murray Valley encephalitis, or a closely related virus, had infected over 30% of wild birds, and that fowls of known age must have been infected during the fifteen months before December, 1954. The experimental evidence that dengue does not infect either fowls or pigeons is quite good, and the serological relation between dengue and Murray Valley encephalitis is unlikely to explain these findings.

If, as now seems probable, there are highly endemic foci of Murray Valley encephalitis in tropical Australia, the presence of such a focus would be another danger which could be revealed by a biological survey of an area scheduled for mineral development, and appropriate action to reduce the mosquito population could be taken. Further, the suggestions of Anderson and Eagle and of Miles and Howes, that exceptionally heavy spring rains in endemic areas presage epidemics of Murray Valley encephalitis, open the possibility that such towns as Broken Hill and Mildura, which have suffered serious outbreaks in the past, may take special anti-mosquito precautions when the spring weather suggests that a spread of the disease is likely.

**Diseases Not Normally Spread by Arthropod Vectors.**

While it is easy to suggest ways of combating those zoonoses spread by arthropod vectors, the problem of the two diseases I now want to discuss is much more difficult They are "Q" fever and psittacosis.

"Q" fever, originally described by Derrick from an outbreak in Brisbane abattoirs, has proved to occur in most countries where a search for it has been made. It has been shown to infect a wide variety of ticks, a variety of wild mammals and also pigeons, but the cycles in which it infects man do not normally involve an arthropod vector. Because of certain morphological and serological differences, and because it can be spread between mammalian hosts without the intervention of an arthropod vector, the rickettsia of "Q" fever has been placed in a separate genus and is named *Coxiella burneti*.

Most human cases of this disease in Australia have been in abattoir workers, and particularly those working on cattle, and it has been thought that the endemic foci in cattle would be found in the dairying areas close to the coast. In our expedition to the Northern Territory in 1952, we found serological evidence of infection among aborigines at Hermannsburg and Haast's Bluff; and on our visit to the same area in 1954 we found that some of the cattle on the Hermannsburg run were also seropositive. Also in 1954 we had for the first time an outbreak of "Q" fever in the mutton hall at the Adelaide abattoirs; and more recently Dane has investigated an outbreak of the disease on a sheep station in the Flinders Ranges and has isolated the organism from crutchings from ewes recently lambed and nearly due to lamb. Infection, therefore, has been proved to occur in sheep as well as cattle in South Australia.

In Australia there is no very good evidence of spread by milk, which appears to be the normal method of spread in South California, but rather the spread would appear to be either in dust, from contaminated ground or wool, or from aerosols from infected tissues set up during the killing of beasts and processing of carcasses in abattoirs.

The percentage of infected animals is probably small, and the distribution of the disease in sheep and cattle in Australia is practically unknown. Avoidance of the possibility of infection among abattoir workers is impossible.

The vaccination of laboratory workers against "Q" fever has been fairly successful, and it would seem that the only method likely to be effective for the control of the disease in Australia is the immunization of the group particularly at risk—the abattoir workers.

By contrast with "Q" fever, which seems to infect only a low percentage of susceptible domestic animals, psittacosis virus (and I now include ornithosis in the same term) infects a very high percentage of wild and domestic birds in Australia. The position in wild and pet-shop psittacine birds was studied by Burnet in 1934, and these studies have been extended more recently in Adelaide (Beech and Miles, 1953; Dane and Beech, 1955). Burnet's findings showed a much higher infection rate among birds bought from dealers than among wild psittacines, and our work has shown that about 80% of homing pigeons and about 50% of domestic fowl are infected in South Australia. The percentage of feral pigeons infected is much lower.

The infection rate among wild birds is very variable, and several common species, especially of non-psittacine birds, of which reasonably long series have been examined, have failed to give any evidence of infections.

Dane (1955) has found that approximately 25% of adults in South Australia have antibodies against psittacosis group viruses, but the nature of the virus stimulating these antibodies is not clear, and certainly most of them must be in response to subclinical or mild upper respiratory infections. There was no clear relation between contact with birds and possession of antibody.

Certainly the rate of notification is low compared with the number of clinical cases of psittacosis which occur in Australia; nevertheless, in view of the apparent opportunities for infection, the number of cases must be very small.

The only possible explanation is that the strains of virus common in Australia are of a low virulence to man. On whether the numerous inapparent human infections with viruses of the group are due to an unknown human virus or to avian viruses, there is at present no evidence.

All the Australian strains of psittacosis viruses so far isolated have been of only moderate virulence for mice when inoculated by the intraperitoneal route. Normally the strains responsible for serious outbreaks of human disease have had a high virulence by this route, and this characteristic of the Australian strains of virus may be related to the lack of large outbreaks in man.

Birds carrying psittacosis virus do not shed the virus all the time; but when they are subjected to any physiological stress, they commonly begin to shed virus. The introduction of new birds in poor condition to a pet shop often leads to an outbreak of the disease, with rapid bird-to-bird passage and possibly exaltation of virulence.

Such pet-shop epizootics lead to outbreaks of human disease; and undoubtedly veterinary control of conditions in pet shops, regulations on the isolation of new consignments of birds, and regular veterinary inspection of birds sent interstate or overseas would reduce both losses to dealers from this disease and the risk of human outbreaks.

In this paper I have discussed the control of some of the more important zoonoses due to viruses and rickettsia. In the case of those normally transmitted by arthropod vectors much can be done by the use of insecticides and the clearing of highly endemic areas of scrub. A strong case can be made, particularly in tropical areas, for a biological survey of an area about to be opened up for the exploitation of minerals or for close settlement, so that the local risks may be known and all possible measures to counteract them taken.

In the case of diseases against which an effective vaccine is available, the immunization of individuals specially at risk may be well worth while. In the special case of psittacosis, regular veterinary inspection of pet shops and of consignments of birds being moved interstate or overseas might considerably reduce the number of infections.

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## CONTROL OF INFECTIOUS DISEASE IN A RURAL COMMUNITY.[1]

By CLIFFORD JUNGFER,
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THE country doctor has many opportunities for the study of infectious disease. He lives in close contact with the community, so that he can appreciate the importance of the human factor in prevention. He can follow the ever-changing pattern presented by even the common infections. In South Australia he is usually the local medical officer of health, and as such he is responsible for initiating measures for the control of infectious disease. These opportunities soon make him realize that our methods need constant revision if they are to remain effective.

When I first entered practice, nearly thirty years ago, an occasional case of typhoid fever was not uncommon in country districts. But during the last twenty years no case of typhoid has occurred in the Adelaide hills. This is not because of better sanitation, as this has not improved throughout the years. That the opportunity still exists for the spread of such diseases is shown by the epidemics of diarrhœa which occur during the summer months. An obvious explanation is that a carrier has not entered the community. This may be chance only; or is it because these reservoirs of infection are recognized and controlled more effectively nowadays?

It is important to remember, however, that rural communities are vulnerable to such diseases as typhoid, and that their vulnerability increases as their population grows and their people travel more widely. What practical measures can we take to safeguard such communities? The mass methods of sanitation which protect cities and towns cannot be applied to our closely settled rural communities. We must rely on two methods. The first is better sanitation of places where people congregate. This will offer some protection against the strangers who visit us. Secondly, by educating the householder, in the villages and on the farms, as to his responsibility, we can widen the margin of safety. In other words, we must rely to a large extent on the cooperation of the community in our efforts for control.

In South Australia recently measles and whooping cough have been removed from the list of notifiable diseases. This indicates that our Health Department realizes that in some instances it is more effective to educate than to legislate. Increased application of this principle should give better results with the control of infectious diseases in rural areas than we have achieved in the past. But removal of the obligation to notify does not mean that we, as doctors, can neglect the community aspects of any of the infectious diseases of childhood. These will always demand our considered attention. They fall into several distinct groups, and sometimes the problem is not only how to control, but how much to control. Some should be completely prevented, whilst others should be controlled to varying degrees.

Measles, mumps and chicken pox, for example, are more safely suffered in childhood unless, of course, the child is very young or sickly, when we can postpone the attack with serum. The severity of whooping cough is diminished, if not prevented altogether, by vaccine. But the position with rubella is rather different. Here we need control effective enough to ensure that this disease is not transmitted to women during the early months of pregnancy. Such a measure of control is difficult unless we can give the adolescent girl the disease. Is this a practical measure now; and if so, how much cooperation can we expect from the community?

Our experience in the Adelaide hills during the last twenty years, with programmes for immunization against diphtheria, gives some information on the extent to which people will cooperate. In 1936 mass immunization cam-

[1] Read at a plenary session on "Control of Infectious Diseases", Australasian Medical Congress (British Medical Association), Ninth Session, Sydney, August, 1955.

paigns were introduced, and as these were well attended we thought that satisfactory group immunity would result. Our complacency was shattered in 1942 by the occurrence of 32 cases of diphtheria with two deaths.

In order to assess the position all children (1040) living in one district were Schick tested. It was found that 66% were immune. Practically all the 34% who were not immune had not been immunized despite very active propaganda.

We then stopped mass campaigns and publicity methods. Instead, a nurse working in the area with the Adelaide Hills Children's Health Survey was placed in charge of a continuous programme, which involved personal canvass of mothers, immunizing the children and regular checking by means of Schick testing. From this time until 1948, when the survey finished, the level of immunity was maintained at 96%. It was hoped that after these five years of intensive and personal education of the mothers, the necessity for immunization had become part of the fixed beliefs of the community.

So since 1948 the parents have been left to make their own arrangements with the doctors of the area. There has been no organized publicity.

This year a group of children, comprising all those in one area, were Schick tested, and the group immunity is now 73%.

It is obvious that 73% of the children belong to families who accept diphtheria immunization as something essential. They will have their children immunized if mass campaigns are available, or, if not, they will make their own arrangements. The remaining 27% represent our problem in health education. They are not susceptible to publicity methods, nor do they show individual initiative, but they will accept passively a personal service for their children which is delivered on their doorstep.

Our next step will be to see whether the group which is "teachable" can help in altering the attitude of the "unteachables".

The 73% group has shown a strong sense of personal responsibility. It is hoped that, when this group understands the problem as it affects the community, they will accept responsibility for raising the level of cooperation in the 23% group.

Another difficult and interesting problem is that presented by the clinical conditions due to infections with hæmolytic streptococci and staphylococci. As an example, scarlet fever has been on our list of notifiable diseases for many years in South Australia. But today we rarely see the florid cases which were common thirty years ago. Perhaps "scarlet fever syndrome" would be a more appropriate description. Are we paying sufficient attention to the widespread activities of these organisms in our community? Over the years I have noticed that when the uneasy balance between the community and these organisms is upset, the clinical manifestations are very varied. Quite often the first indication of trouble in the district is given by a small group of children who present with throat infections, some may show a fleeting rash, and then follows a trail of other patients. These show diverse clinical conditions, such as skin infections, with occasional erysipelas in an aged patient, *otitis media*, adenitis and even acute rheumatism and acute nephritis.

Although this pattern is well recognized, puerperal fever and wound infections still occur in our hospitals, but we feel safe because treatment with modern drugs is usually effective.

But is our position as secure as we imagine it to be? Can we claim that the actual incidence of these conditions is less now than it was in the days when rigid methods for prevention were our only safeguard.

The resistant strains which are arising are a warning, surely, that the organisms we feared in the past merit at least our respect in the future.

And, finally, if it is accepted that our methods for controlling infectious diseases need revision to make them suitable for the future, may I presume to speak on behalf of Australian country practitioners? There are three ways in which we can receive help from our colleagues in Health Departments, in order that our methods for the control of infectious disease can meet the ever changing need:

1. By provision of bacteriological services, which are readily available and independent of financial considerations. Bacteriological investigations have always been an essential part of any programme for the control of infectious disease. In these days when antibiotics modify the behaviour of the infecting organisms these facilities become even more important. They are necessary for the general practitioner who wishes to study infectious disease in all its aspects in his community. If we are to inspire our young graduates with the vision of a Pickles, we must show them that, although most of the alluvial deposits of general practitioner research have been well explored, there still remain reefs with payable ore in them. To work these reefs will require modern tools, which we should be willing to give them.

2. A more rapid and effective method should be devised for the exchange of information concerning the type of infectious disease occurring in various parts of the country. Formal notification alone does not achieve this. The practitioner is interested in the clinical features of the infectious diseases which are affecting other communities. This is particularly so when the pattern is unusual, as it so often is in the early stages of an outbreak. With this knowledge he can provide earlier diagnosis, and this may lead to more rapid control of the infection. With his help the community may continue the action.

3. And lastly, in any plans for the better integration of the health service, it should be recognized that the general practitioner lives and works at the level at which good planning most frequently bursts forth into effective action.

May I conclude by quoting Sir Macfarlane Burnet, whose writings on the biological aspects of infectious disease give us so much inspiration:

> Infectious disease is, and always has been, part of the every day experience of life. In every generation men of affairs have had to cope as best they could with the practical problems it presents, while priests, philosophers and, later, scientists have had perhaps the harder task of interpreting the significance of such disease in accordance with the intellectual outlook of their time.

## THE TREATMENT OF ULCERATIVE COLITIS.

By E. S. R. HUGHES,
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EFFORTS to find a specific cure for ulcerative colitis have not, so far, been rewarded, and the relatively high mortality and morbidity still associated with the disease are disappointing.

Rice-Oxley and Truelove (1950) found, after an initially high fatality rate of 22% in the first year, that the dangers associated with ulcerative colitis diminished; but they noted that another 10% of patients were likely to die before five years had elapsed. Joske (1952) studied 99 unselected patients admitted to the Royal Melbourne Hospital from 1936 to 1944 and followed to 1949; of this series, 25 died in hospital and four others died subsequently from the disease. Wheelock and Warren (1955) reviewed the fate of 343 patients admitted to the Massachusetts General Hospital before 1943, and traced for ten years or until death; of these, 188 (54·7%) were dead and 153 (44·6%) had died from ulcerative colitis.

The high mortality rate in the early stages of the disease is due to toxæmia, hæmorrhage, perforation and abscess formation, and a fatal outcome is not uncommon within a few weeks of the onset of the first symptom. Joske (1952), in the Royal Melbourne Hospital series, made the important observation that the outstanding feature of the autopsies was the large number of complications present in each case. In fact, this was so notable that it appeared likely that such complications were present, but not diagnosed, in the patients before their death.

FIGURE I.

Mrs. R., aged forty-two years, had suffered from "colitis" for fourteen years; in the last four months she had experienced severe abdominal colic. Laparotomy revealed ulcerative colitis, and a carcinoma of the hepatic flexure. A one-stage colectomy and ileo-rectal anastomosis was performed. She remained very well for six months, but then quickly succumbed with peritoneal metastases.

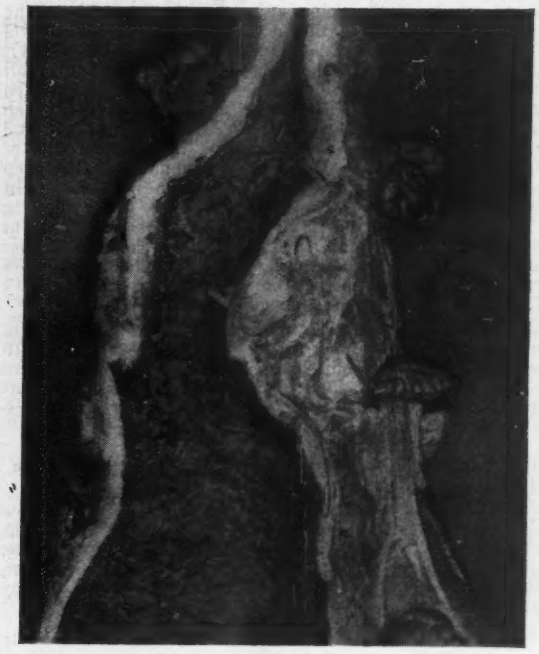

FIGURE IIB.

A closer view of the same specimen, to show that some distensibility is retained by the caecum.

FIGURE IIA.

Mrs. F., aged fifty-five years; one-stage proctocolectomy for ulcerative colitis. There is gross fibrosis around the rectum.

FIGUURE IIIA.

Miss G.; ileostomy, followed by one-stage proctocolectomy. The bowel is moderately contracted.

It has become accepted by nearly all authorities that the risk of cancer of the colon and rectum is greater in the presence of ulcerative colitis than in the normal bowel (MacDougall, 1954; Cuthbert Dukes, 1954). Zetzel (1954), after reviewing current opinions, observes that 3% of all patients with colitis develop cancer; 11% of those treated surgically, and 20% of those with colitis of more than twenty years' duration, develop cancer. Wheelock and Warren (1955) stress the fact that there is no way of predicting which patient develops carcinoma, and that even those with mild disease are more prone to malignant disease than patients with normal colons. After making an analysis of 1500 cases, Bargen and his associates at the Mayo Clinic (Bargen *et alii*, 1954) concluded that carcinoma of the large bowel was 20 to 30 times more common as a cause of death among patients with ulcerative colitis than in the population as a whole (Figure I).

Because of this improved knowledge of the natural course of this disease, and because of better surgical facilities, there has, in recent years, been a considerable trend towards surgery. In such circumstances, it is important that treatment be continually subject to review if unnecessary operation is to be avoided.

### Place of Psychotherapy in the Treatment of Ulcerative Colitis.

Over the past twenty-five years, emotional disturbances and personality traits have been regarded as important factors in the development of ulcerative colitis. Grace, Wolf and Wolff (1951) have summarized their work on this aspect in an excellent and painstaking report. They make the following statement:

> It appears, therefore, that the colon of man is likely to participate in his reaction patterns to threatening events in his life situation, including those threats which arise out of problems of interpersonal adjustment. It further appears that changes involving colonic hyperfunction, at first altogether functional and transitory, may, when unduly sustained, result in structural damage and disease . . . The patient with ulcerative colitis was found to be characteristically an outwardly calm, superficially peaceful individual of more than usual dependence. On going beneath this calm exterior, it became apparent that this outwardly placid person is "sitting on a powder keg" of intense hostility, resentment and guilt. These long-standing, unrelieved feelings were associated with hyperfunction of the colon, with increased motor activity, increased vascularity, turgescence and small hæmorrhagic lesions.

The failure of a disease, such as ulcerative colitis, to respond to non-operative treatment is exasperating, and an explanation is often sought in the patient's mental attitude. In the writer's opinion, any peculiarities of personality in this disease are the sequel of the incessant diarrhœa, rather than a cause of it. A personal and regular review of 18 patients with ileostomy has failed to reveal any abnormality in personality, an experience similar to that of Counsell and Lockhart-Mummery (1954); the latter also suggested that too much emphasis might have been placed on the emotional factor in the ætiology of this disease.

The more resistant an individual to treatment in this disease, the less the indication for psychotherapy. These patients should be treated as normally reacting people. The disease should be explained to them; they should be reassured that there is no malignant disease; they should be told that they can be cured, but that this may mean operation.

### Place of Medicine in the Treatment of Ulcerative Colitis.

If the nature of this disease is explained to the patient, and if he is given advice as to how to maintain his general condition at an optimum level, the need for special measures is often averted.

In the more severe cases in which there is considerable loss of protein and blood from the large bowel, admission to hospital should not be delayed. Complete rest in bed, combined with long hours of sleep at night, a protein-rich diet with added vitamins, and transfusion of whole blood may be rewarded by remission of symptoms and reduction in the severity of the disease.

Rice-Oxley and Truelove (1950) found that penicillin and non-absorbable sulphonamides made no difference to the course of ulcerative colitis. Zetzel (1954) observed that the inconstant response of uncomplicated, though active, colitis to chemotherapeutic agents has been a disappointing climax to the enthusiasm that greeted their first appearance.

There is some difference of opinion concerning cortisone and ACTH. Truelove and Witts (1954) organized an extensive trial in the United Kingdom, and in a progress report stated that cortisone improved the well-being of the patient, as well as favourably influencing the sigmoido-

FIGURE IIIB.

A closer view of the same specimen to show the sigmoid flexure; note the small ulcers.

scopic and radiological findings. However, they found that relapse was common after cessation of treatment, and they stated that their figures showed no statistical lessening of the need for ileostomy or in the mortality from the disease. At the time of this report, it was not possible to foretell whether or not cortisone would have any lasting benefit in the natural history of the disease. Zetzel (1954), after reviewing the literature, reached a similar conclusion; there was no denying the powerful properties of these drugs, and there seems to be little doubt that their administration induces a clinical remission in some cases; but there is no evidence to show that the drug is curative, and certainly it should not be used indiscriminately.

### Place of Surgery in the Treatment of Ulcerative Colitis.

The present trend towards surgery is revealed in the figures of Cattell and Colcock (1955); formerly only 25% of patients were subjected to operation, but the latest figures indicate that at the Lahey Clinic 50% are now subjected to operation. With this increase in surgical treatment there has been a considerable fall in the overall mortality from the disease.

When operation is necessary, an ileostomy is nearly always unavoidable; and in the majority of patients who

are given an ileostomy, it will prove to be permanent. However, the ileostomy is not the impossible handicap that has been visualized by some. In not a single case, amongst the writer's patients with ileostomy, has there been anything more than a temporary inconvenience. Counsell and Lockhart-Mummery (1954) investigated the lives of 52 patients with an ileostomy. They found that these patients were healthy, without exception; they possessed

FIGURE IIIc

A close view of the specimen to show the transverse colon; note the small ulcers.

normal personalities, and were active and independent, and were well able to cope with life's difficulties, in spite of the handicap; practically all returned to normal work; they were able to engage in normal sporting and social activities; they were able to eat anything well chewed, provided the food did not have malodorous after-effects; sexual activity returned to normal. One of the writer's patients is a schoolboy, aged fourteen years, and he takes part in normal school life and sporting activities. Two of the females have since married, and one has had a baby. The life of the ileostomy patient has been facilitated considerably by the use of improved ileostomy bags. Special attention to the ileostomy stoma has also been responsible for greater freedom from complications which have marred the life of the ileostomy patient in the past. In the writer's series, a split skin graft has been applied to the ileostomy on 15 occasions, but the procedure has proved disappointing because of the high incidence of ileostomy stenosis needing correction (Hughes, 1956, in the press).

It is impossible to save the colon in those patients with ulcerative colitis severe enough to need an ileostomy. Attempts have been made to close the ileostomy; but Cattell (1953) reports that approximately half the patients who underwent closure of the ileostomy had recurrence of symptoms, necessitating a second operation within five years. Furthermore, the risk of cancer is formidable.

Opinion is not so well defined on the question of preservation of the rectum. The clinician is reluctant to advise a permanent ileostomy, and since he is able to keep the rectum under observation with the sigmoidoscope, he may decide in favour of joining the ileum to the rectum (Aylett, 1953, 1955). However, in the hands of most surgeons, the operation has a definite morbidity and mortality, whilst the danger that carcinoma may develop in the rectal stump, despite its accessibility to clinical examination, is very real.

The orthodox method of performing excision of the large bowel involves two or three stages. Cattell (1953) favours three stages; an ileostomy is followed at intervals of three months by colectomy and subsequently excision of the rectum. Gardner and Miller (1951) reported a series of 17 cases of fulminating ulcerative colitis in which ileostomy with primary colectomy was performed at the initial operation; this procedure has received support from others (Ripstein, 1953; Goligher, 1953; Avery-Jones, 1953).

In selected cases, and in certain circumstances, the rectum and colon can be excised at the same time as the ileostomy is constructed (Hughes and King, 1955). This

FIGURE IIId.

A close view of the specimen to show the caecum; again note the numerous small ulcers.

is not the formidable operation that it seems. Goligher (1954) reported 14 cases in which this method was used, including two in which the patients were gravely ill, and was impressed by the smooth recovery of the patients. The writer has performed 10 procto-colectomies, and eight of them have been performed at the time of construction of the ileostomy (Figures IIA and IIB and IIIA, IIIB, IIIC and IIID). The operation presents no difficulties. There have been two deaths in the writer's series; in the remaining eight, the course has been uneventful from the commencement of the operation. One patient died from pulmonary embolism on the twelfth post-operative day,

after a seemingly uneventful convalescence. The second, a woman, aged sixty-two years, was almost moribund at the time of the operation, at which free gas was found in the peritoneal cavity, associated with multiple perforations in the colon from the caecum to the rectum. This patient was not disturbed by the operation, and she appeared set for a good recovery, when she died suddenly on the second day after operation. She had been treated with cortisone prior to operation, and there was considerable œdema of the tissues and especially the lungs.

### Summary.

1. The natural history of ulcerative colitis is now better known than previously. There is a relatively high mortality in the first twelve months of the disease. Relapses are common, and the longer the patient is followed, the more likely he is to suffer a relapse. In long-standing cases there is a real risk of the development of cancer.

2. Psychotherapy has been used over the past twenty-five years, and many clinicians believe that it has an important role in the treatment of this disease. However, some believe that psychogenic manifestations follow the disease, and support for this is to be found in the normal behaviour of those in whom an ileostomy has been established. Attempts to psychoanalyse patients with this disease may be responsible for unfortunate delays in instituting sounder treatment.

3. Non-operative treatment may control the disease. Rest, adequate sleep, attention to diet and vitamin intake, and blood transfusions are the most important of these non-operative measures. Chemotherapy, including the administration of antibiotics, cortisone and ACTH, should be used cautiously and with the knowledge that the value of these agents has yet to be proved.

4. More patients with this disease are now being subjected to operation. This has been the direct result of improvements in the management of the ileostomy stoma and the unsatisfactory results of treatment by non-operative methods. Regular reviews of ileostomy patients reveal that very few are incapacitated in any way by the artificial stoma, and all are emphatic that they are much more comfortable with the ileostomy than they were without it. The patient who needs an ileostomy needs a colectomy; most of these patients will require removal of the rectum. Before an ileum is joined to the rectum in this disease, careful consideration must be given to the mortality and morbidity which accompany this procedure. Colectomy with excision of the rectum is usually performed in different stages, but can be performed at the same operation, and has been successfully accomplished at the time when the ileostomy is initially constructed.

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## THE INDUCTION AND CONTROL OF HYPOTHERMIA.

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NUMEROUS reports have appeared in medical literature in recent years on hypothermia—that is, the lowering of body temperature to obtain a significant reduction in metabolism. In general these reports have been concerned more with the depths to which hypothermia could be carried before cardiac arrest or ventricular fibrillation intervened, and with the benefits accruing from its use, than with the relative merits of the various possible methods of inducing the hypothermic state and restoring the normal one.

The results of investigations on the effects and limitations of hypothermia will inevitably be difficult to interpret until good control of the state of hypothermia of the patient can be achieved. In this paper techniques for the induction and control of hypothermia are critically examined, and it is concluded that a method using so-called blankets containing channels through which water can be circulated, which can be brought into intimate contact with the patient, has most in its favour. The application of apparatus constructed on this principle to sheep and to a human subject is described.

### Techniques for Hypothermia.

The many different methods that have been used for hypothermia in animal and human subjects are set out in the first column of Table I, in the second column of which representative references are given to descriptions of the techniques used. Important factors to be considered in assessing the relative merits of these various techniques are the following: (i) It should be possible to lower, maintain and subsequently raise the subject's temperature in a controlled manner. (ii) The equipment should be simple and convenient to operate. (iii) There should be as little interference as possible with normal anæsthetic management, antiseptic precautions and surgical access. (iv) Dangers arising from the technique itself should be kept to a minimum; in particular there should be no risk of frostbite or burning, and the subject should be accessible for immediate remedial measures should fibrillation or cardiac arrest occur. (v) The overall time required for the induction of the desired degree of hypothermia and the subsequent rewarming of the subject should be as short as is practicable.

The foregoing characteristics are not wholly independent of one another, and in particular (i), (iv) and (v) are

[1] The work described in this paper was done while the writer was a Research Fellow of The Royal Australasian College of Physicians attached to the Royal North Shore Hospital, Sydney.

TABLE I.

*Techniques for Hypothermia, with Observed Rates of Change of Temperature.*

| Technique for Heating or Cooling Subject. | Reference. | Rate of Change of Temperature (Degrees Centigrade per Hour) and Temperature Overshoot (Degrees Centigrade: in Italics). | | | | |
|---|---|---|---|---|---|---|
| | | Dog. | Monkey. | Sheep. | Child. | Man. |
| "Lytic cocktail" of pethidine, promethazine and chlorpromazine. | Laborit as quoted by Dundee, Gray *et alii* (1953) | — | — | — | — | −0· |
| | Smith and Fairer (1953) | — | — | — | — | — |
| | Shackman *et alii* (1954) | — | — | — | — | 0 |
| | Ripstein *et alii* (1954) | — | — | — | — | −4 |
| Circulation of blood in external coil. | Delorme (1952) | −30 | — | — | — | — |
| Circulation of saline in pleural cavity. | Blades and Pierpont (1954) | −12 | — | — | — | −3, *1* |
| Bath, iced water | Churchill-Davidson *et alii* (1953) | +18 to +24 | — | — | — | +4 |
| | Swan *et alii* (1953) | −11, *2 to 3* | — | — | — | — |
| | | — | — | — | −6 to −33, *2 to 8* | −6 to −8, *7* |
| | Callaghan *et alii* (1954) | — | −10 to −30, *2* | — | — | — |
| | Edwards *et alii* (1954) | −3 to −4 | — | — | — | — |
| | Lynn *et alii* (1954) | — | — | — | — | — |
| | Swan and Zeavin (1954) | — | — | — | — | — |
| | This paper | — | — | −13, *2* | — | — |
| Bath, warm water | Bigelow, Lindsay, Harrison *et alii* (1950) | +8 | — | — | — | — |
| | Callaghan *et alii* (1954) | +9 to +25 | — | — | — | — |
| | Fleming (1954) | +9 | — | — | — | — |
| | Lewis *et alii* (1954) | — | — | — | +10 to +17 | +6 to +10 |
| Ice packs | Smith and Fay (1940) | — | — | — | — | — |
| Ice packs with chlorpromazine | Dundee, Gray *et alii* (1953) | — | — | — | — | −2, *2* |
| | Dundee, Scott and Mesham (1953) | −7 | — | — | — | — |
| | Bigelow *et alii* (1954) | — | — | — | — | — |
| Cold or warm air | Bigelow, Lindsay and Greenwood (1950) | — | — | — | — | — |
| | Bigelow, Lindsay, Harrison *et alii* (1950) | −6 | — | — | — | — |
| | Cookson *et alii* (1952) | −3 to −4 | — | — | — | — |
| Electric blanket heating | Cookson *et alii* (1952) | +4 | — | — | — | — |
| Radio-frequency heating | Bigelow, Callaghan and Hopps (1950) | +3 to +13 | — | — | — | — |
| | Bigelow *et alii* (1954) | — | — | — | — | — |
| | Callaghan *et alii* (1954) | — | +9 to +25 | — | — | — |
| Large "blankets" for circulation of liquid. | Bigelow, Callaghan and Hopps (1950) | — | — | — | — | — |
| | Bigelow and McBurnie (1953) | — | −18 to −36 | — | — | — |
| | Fleming (1954) | −4 | — | — | — | — |
| | Inglis *et alii* (1954) | — | — | — | −10 | — |
| | Lewis *et alii* (1954) | — | — | — | −4 to −5, *3* | −2 to −4, *3* |
| | Pontius *et alii* (1954) | — | — | — | — | — |
| | Ripstein *et alii* (1954) | — | — | — | — | −1 |
| As above, with chlorpromazine | Bigelow *et alii* (1954) | — | — | — | — | — |
| | Ripstein *et alii* (1954) | — | — | — | — | −2 |
| | | — | — | — | — | +2 |
| Set of "blankets" for circulation of liquid with chlorpromazine. | This paper | — | — | −3, *0·3 to 1* +3 | — | −4, *0·7* +3 |

linked. This is because a technique for which the rate of heat transfer is high, and for which the cooling and heating of the subject are therefore rapid, is likely as a consequence to provide poor control of the subject's temperature and to be prone to a considerable overshoot of temperature after cooling has been stopped. Such an overshoot constitutes a serious danger in the event of fibrillation or cardiac arrest occurring during cooling. A summary is given in Table I of the rather sparse information available on the actual rates of cooling and warming and of the temperature overshoot observed with the various techniques. The figures confirm, in a general way, that high rates of changes of temperature result from methods which inherently provide a high rate of heat transfer, though some figures are clearly anomalous, presumably because of features of the actual technique used. The figures for temperature overshoot, given in italics in the table, confirm that this can be a serious problem.

Since it would seem that the subject's welfare is not significantly affected by the length of time spent in the hypothermic state (Smith and Fay, 1940; Bigelow, Lindsay, Harrison *et alii*, 1950; Swan, 1954), it is logical to favour a technique for hypothermia which provides good control of the subject's temperature at all stages, even if this means using low rates of change of temperature and hence lengthening the overall time required for cooling and rewarming the subject. With some methods it is possible to offset the increases in these times by overlapping the late stages of the cooling and the commencement of the rewarming with the time required for operation.

A consideration of the various techniques of Table I, made in terms of the factors listed above, is summarized in Table II. In this table the heading "Ease of Application" is meant to relate to the difficulties inherent in the actual application of the method, rather than to the difficulty of obtaining or setting up the equipment. With regard to the latter, it is unfortunate that the "lytic cocktail", bath, and ice-pack methods, which require the simplest equipment, are unsatisfactory from the point of view of temperature control.

Certain of the methods listed have additional features which should be taken into account in assessing them. These are discussed below.

It would seem that the "lytic cocktail" alone cannot be considered to induce true hypothermia (Shackman *et alii*, 1954), since the drop in temperature, if any, is at most 4° C. (Ripstein *et alii*, 1954). However, it has been used extensively as an adjunct to other methods.

The by-passing of part of the subject's circulation through an external coil requires interruption of the continuity of one or two large blood vessels. This, together with the dangers of hæmolysis and clotting in the cooling apparatus, excessive bleeding due to measures taken to prevent clotting during hypothermia, and the risks of sepsis and thrombosis in the resutured vessels all tend to make extracorporeal cooling of the blood-stream unattractive. Because of the danger of ventricular fibrillation, excessive cooling of the heart must be avoided with this method, as with cooling of the open pleural cavity with saline.

The bath technique provides rapid cooling, but, as was indicated above, this accentuates the dangers arising from cardiac arrest or fibrillation; not only is the temperature overshoot likely to be large, but the time required to prepare the patient for cardiac massage will also be greater than for most other methods. The use of a bath for rewarming the subject involves problems of asepsis, particularly if a pleural drainage tube is present.

TABLE II.

*Characteristics of Techniques for Hypothermia.*

| Method of Cooling or Rewarming Subject. | Control of Rate of Change of Temperature. | | | Ease of Application. | Anæsthetic Management. | Asepsis. | Surgical Access. | |
|---|---|---|---|---|---|---|---|---|
| | During Cooling. | During Operation. | During Rewarming. | | | | During Cooling. | During Operation. |
| "Lytic cocktail" .. | Poor. | Poor. | Poor. | Good. | Good. | Good. | Good. | Good. |
| Circulation of blood in external coil .. .. | Good. | Good. | Good. | Difficult. | Good. | Difficult. | Good. | Good. |
| Circulation of saline in pleural cavity .. .. | Good. | None. | Good. | Limited to thoracic surgery. | Good. | Difficult. | Excellent. | Good. |
| Bath, iced water .. | Very poor. | None. | —— | Fair. | Difficult. | Poor. | Poor. | Good. |
| Bath, warm water .. | —— | None. | Poor. | Fair. | Difficult. | Poor. | —— | —— |
| Ice packs .. .. | Fair. | Poor. | —— | Good. | Good. | Good. | Good. | Good. |
| Cold or warm air .. | Good. | None. | Good. | Fair. | Fair. | Good. | Fair. | Good. |
| Electric blanket .. | —— | —— | Good. | Good. | Good. | Good. | —— | —— |
| Radio-frequency heating | —— | —— | Good. | Good. | Good. | Good. | —— | —— |
| Large "blankets" for circulation of liquid .. | Good. | Poor. | Good. | Fair. | Good. | Good. | Fair. | Good. |
| Set of "blankets" for circulation of liquid .. | Good. | Good. | Good. | Fair. | Good. | Good. | Good. | Good. |

The circulation of cold air past the subject appears to have been used only on animals. The rate of cooling obtained with dogs by the use of an air temperature well below 0° C. was only of the order of 6° C. per hour; that with an adult human being would be considerably less than this. The use of sub-zero air temperatures means that extreme care must be taken if frostbite is to be avoided.

The electric blanket is simple, and with reasonable care provides a safe method of rewarming the subject. A number of individual blankets would facilitate the commencement of warming before the completion of an operation.

The use of so-called blankets in which a liquid circulates has the advantage that the subject need not be moved from the operating table from the commencement of the cooling till the completion of the rewarming. The blankets hitherto described have consisted of one or two units, somewhat resembling electric blankets, except that rubber or plastic tubes have replaced the electric elements. The actual area of contact between the tubes and the skin is inevitably small, leading to a correspondingly lower rate of heat transfer between the skin and blankets than with the bath technique, especially if the use of a cooling liquid below 0° C. is avoided to preclude frostbite. With large blankets it is difficult to maintain a controlled degree of hypothermia during an operation, because of the even poorer thermal contact with the subject's skin after portion of the blanket has been unwrapped to provide surgical access. These disadvantages are obviated to a large extent by using a set of suitably designed smaller blankets, portion of only one of which need be removed to provide surgical access.

### Technique Selected for Use.

In the light of the foregoing assessment it was concluded that, of the techniques examined, the most satisfactory for the cooling and subsequent warming of human patients in connexion with the application of hypothermia for surgical purposes is to use a set of suitable blankets through which water at an appropriate temperature can be circulated. Those developed for this purpose were five in all, as set out in Table III. They fitted fairly closely on an average adult, thus providing better contact with the patient's skin than would be possible by the use of large blankets. By unwrapping the thoracic blankets but leaving them under the patient access may be gained to the chest without disturbing the heat-transfer arrangements over the rest of the body. In this way it was hoped that it would be possible to maintain adequate control of the hypothermic state while the operation was in progress, and that effective rewarming could be started before closure of the surgical incision. In each blanket the water flow is through a continuous channel of half-inch by quarter-inch cross section formed by the gaps between rubber septa projecting alternately from either end of the blanket to near the other end, and bounded top and bottom by rubber sheets, three thirty-seconds of an inch thick, and vulcanized onto the septa, which are one-quarter of an inch thick and three-eighths of an inch wide.

Rubber tubing was used to connect the blankets to a manifold into which water was pumped from a tank of about four gallons' capacity, containing ice and water. After flowing through the blankets, which were connected in parallel, the water returned to the tank. Adjustment could be made of the relative rates of flow through the blankets by clips on the rubber tubes leading to them, and of the overall circulation by a by-pass from the pump back into the tank. To change the temperature of the water it was a simple matter to divert the blanket outlets to a drain and replenish the tank with hot water or ice as required.

TABLE III.

*Set of "Blankets" for Hypothermia.*

| Location. | Size. | Number. |
|---|---|---|
| Thigh, groin to knee .. .. .. | 2 feet by 1 foot. | 2 |
| Lower section of trunk, lower costal margin to inguinal regions. | 4 feet by 1 foot 3 inches. | 1 |
| Thorax, axilla to lower costal margin | 4 feet by 9 inches. | 1 |
| Shoulders, back of neck, upper part of thorax, arms. | 4 feet 6 inches by 6 inches. | 1 |

In the applications described below, ice was used rather than a refrigerator as the means of cooling the water, because it more easily met the considerable refrigeration load involved (up to 1,000,000 calories). Water at about 40° C. was used for rewarming the subjects. For the earlier cases the temperature was controlled by the addition of hot water to the tank, and for the later cases by the adjustment of the power supplied to an immersion heater.

The temperatures of the blankets and of various parts of the subject's body were measured with copper-constantan thermocouples and were recorded on a self-balancing potentiometer. The temperatures so measured were accurate to about ± 0·2° C. Rectal temperatures accurate to ± 0·1° C. were measured with a direct-reading resistance thermometer.

The cooling of a subject with blankets may be assisted to some extent by the use of ice bags. Bags, each about eighteen inches long, of polyethylene tubing of eighteen inches' circumference, filled with a mush of shaved ice and water and sealed at each end with a rubber band, were found to be very convenient for this purpose.

### Hypothermia with Sheep.

Closely shorn sheep were used to test the equipment, since they could be expected, on account of their size, to give a good indication of the cooling rates likely with adult human patients.

TABLE IV.
*Summary of Results.*

| Case. | Method of Cooling. | Cooling Rate. (Degrees Centigrade per Hour.) | Stabilization. | | | Rewarming Rate. (Degrees Centigrade per Hour.) | Minimum Temperature for Natural Respiration. | Chlorpromazine (Total Dose). (Milligrammes.) | Wool Length. (Inches.) |
|---|---|---|---|---|---|---|---|---|---|
| | | | Commenced. (Degrees Centigrade.) | Attained. (Degrees Centigrade.) | Time Required. (Minutes.) | | | | |
| Sheep I .. | "Blankets." | 2·8 | 29·8 | 28·9[1] | 25[1] | 1·9 | — | 100 | 0·5 (wet with castor oil). |
| Sheep II .. | "Blankets." | 3·2 | 26·6[1] | 26·1[2] | 15[2] | — | — | — | — |
| | | 1·3 | 23·9 | 23·6 | 10 | 3·4 | 25·5 | 450 | 0·25 |
| Sheep III .. | "Blankets." | 2·8 | 23·6[1 2] | 22·5[1 2] | 35[1 2] | — | — | — | — |
| | | 0·7 | 22·0 | 21·5 | 15 | 2·7 | 24·6 | 275 | 0·25 |
| Sheep IV .. | "Blankets" *plus* ice bags. | 4·8 | 28·4 | 28·0 | 15 | 2·3 | — | 200 | 0·1 |
| Sheep V .. | Bath. | 12·9 | 30·0 | 27·5 | 30 | — | — | 50 | 0·1 |
| Adult human | "Blankets" *plus* ice bags. | 3·6 | 31·1[1] | 29·2[1] | 25[1] | 2·5 | — | 50 | — |

[1] Temperature allowed to drift to the stabilization value.
[2] Temporary stabilization of temperature followed by further cooling.

Atropine, one-quarter grain, was given thirty minutes before the induction of anæsthesia with sufficient pentobarbitone (60 milligrammes per millilitre) given intravenously to allow the insertion of a polyethylene cannula into the jugular vein and a cuffed Magill's tube into the trachea. Subsequent maintenance doses of one to two millilitres of pentobarbitone solution were given as required to stop movement. The endotracheal tube was connected to a closed circuit with a carbon dioxide absorber, and oxygen was supplied through a modified oxygen-demand valve.

The rectal thermometer was inserted and waterproof thermocouples were sutured into small skin incisions in the neck, on both sides of the trunk, on one leg near the groin, and just above the hoof of one hind leg.

The blankets, which had been placed on the table before the sheep, were then wrapped so that one covered the pelvis and buttocks, a second encircled the trunk between fore and hind legs, while a third covered the shoulders.

Four sheep were cooled in this fashion. The results of these experiments are summarized in Table IV and are shown graphically for one of the sheep (number III) in Figure I.

For sheep I the circulating water was cooled with a commercial water cooler, but at no time was the water temperature below 11° C. In all subsequent experiments and for the patient, ice, added to the reservoir, was used in place of the cooler. In this way water temperatures of about 4° C. were easily maintained during cooling.

As sheep I shivered throughout the period of the experiment, sheep II was given more chlorpromazine (intravenously), the onset of shivering being taken as the indication for a further dose. The quantity of pentobarbitone required to keep sheep II immobile was, as a consequence, somewhat less than for sheep I. Both sheep recovered satisfactorily, even though the rewarming of sheep II was stopped at 34° C. (the normal temperature of a sheep is about 38° C.).

To test the effect of pentobarbitone alone in controlling shivering, no chlorpromazine was given to sheep III at first; but despite very high dosage of pentobarbitone (over three grammes in 3·3 hours) it proved impossible to prevent shivering. Chlorpromazine quickly controlled the shivering and increased the rate of cooling, as can be seen in Figure I. When the temperature of the sheep was 22° C. the large blanket around the sheep's trunk developed a defect, and the pressure of the resulting bulge on the abdomen caused the sheep to regurgitate stomach contents. The blanket was replaced by two small ones and warming was commenced. Unfortunately the hot-water supply failed when the rectal temperature was only 32·8° C. The sheep was still well anæsthetized and not shivering, because pentobarbitone and chlorpromazine had been given in anticipation of a long period of rewarming. With the removal of the Magill's tube a large amount of vomitus was removed from the pharynx, and when the sheep was placed in the pen an hour later it was still unconscious and was not shivering. The sheep died during the night.

Sheep IV was very closely clipped, and in addition to the cooling blankets polyethylene bags filled with crushed ice were packed in spaces between the blankets and the sheep. The rate of cooling for this sheep was appreciably greater than in the earlier experiments.

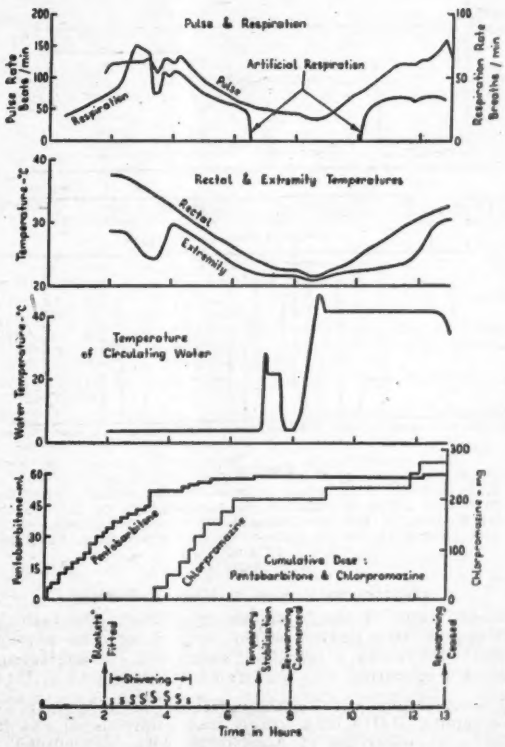


FIGURE I.
Hypothermia with sheep III: rectal temperature, extremity temperature, pulse and respiration in relation to temperature of the water circulating in the blankets and drugs administered, each plotted as a function of time.

In order to obtain figures on the rate of cooling resulting from the use of a bath of iced water and on the temperature overshoot occurring after removal from the bath, tests were made on a further sheep (V). As had been

expected, the rate of cooling was much greater than with the blankets, as was also the temperature overshoot.

From the results of the foregoing experiments it was concluded that a reasonable rate of change of temperature could be expected from the use of the blankets on an adult human being, the more so since (i) the fit of the blankets could be expected to be better than on the sheep, (ii) they would have a relatively larger area of direct contact, and (iii) the insulating effect of the sheep's residual fleece would be absent. The experiments also clearly confirmed that the technique would be capable of giving very good control of the patient's temperature at all stages of the hypothermia.

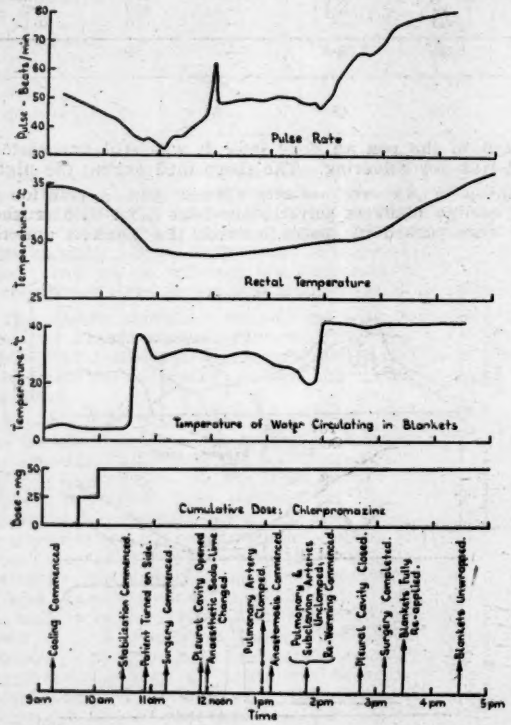


FIGURE II.

Hypothermia with adult human being: rectal temperature and pulse in relation to temperature of the water circulating in the blankets, chlorpromazine administered and stages of the operation, each plotted as a function of time.

## Hypothermia with a Human Patient.

In the light of the foregoing conclusions the technique was applied to a human patient, a man, aged twenty-seven years, with Fallot's tetralogy confirmed by angiography. Blalock's operation was performed on December 9, 1954.

Premedication with pethidine (100 milligrammes) and scopolamine (1/150 of a grain) was administered one hour before the induction of anæsthesia with thiopentone (0·4 gramme), "Eulissin" (three milligrammes) and "Scoline" (75 milligrammes) to allow tracheal intubation at 8.35 a.m. Anæsthesia was maintained throughout with nitrous oxide and oxygen. After anæsthetization, copper-constantan thermocouples enclosed in plastic tubing of two millimetres' diameter were inserted into small stab wounds under the skin of the left thigh, right buttock, right ankle and right wrist. An additional thermocouple was attached to the bulb of a resistance thermometer inserted into the rectum. The five blankets (which were already in position under the naked patient) were applied around each thigh, the pelvis and lower part of the abdomen, the chest and the shoulders respectively. Polyethylene bags filled with ice mush were placed over some parts of the arms and legs not covered by the blankets, and cooling was commenced at 9.15 a.m. with the rectal temperature at 34·7° C. The course of the hypothermia is plotted graphically in Figure II.

At 9.40 a.m. (rectal temperature 34·3° C.) 25 milligrammes of chlorpromazine were given in the intravenous saline drip solution, and a further 25 milligrammes of chlorpromazine were given at 10 a.m. (temperature 33·4° C.). At 10.30 a.m. it was decided to prepare to stabilize the patient's temperature, and by 10.40 a.m., when the rectal temperature was 30·1° C., the temperature of the circulating water had been raised to 37° C., after which the water was brought to a temperature in the vicinity of the patient's temperature. No attempt was made to effect rapid stabilization, but this had effectively occurred by 10.53 a.m. at a temperature of 29·2° C., when the two blankets around the shoulders and chest were unwrapped and the patient was turned on his side. The operation site was prepared, and the skin incised at 11.15 a.m., and five milligrammes of tubarine were administered intravenously. The pleural cavity was opened and the rib spreader was inserted at 11.55 a.m., when the rectal temperature was 28·9° C. The anastomosis was commenced at 1.10 p.m. (rectal temperature 29·4° C.) and was functioning with all clamps removed at 1.50 p.m. During the period from 12.45 to 1.40 p.m. the temperature of the circulating water was progressively dropped from 31° to 20° C. to counteract the upward trend occurring in the patient's temperature. At 1.50 p.m. warming was commenced by raising the water temperature to 42° C., the rectal temperature being 29·9° C. The pleural cavity was closed at 2.43 p.m. when the rectal temperature was 30·5° C. At 3.30 p.m. the patient was laid on his back and all the blankets were reapplied; his temperature was then 32·5° C. At 4.30 p.m. the blankets were unwrapped (rectal temperature 35·1° C.), and by 5 p.m. the patient was awake with a temperature of 36·1° C.

## Discussion.

The rate of cooling obtained with sheep IV was somewhat greater than that with sheep I, II and III in part because the cooling produced by the blankets was in this case supplemented by the use of ice packs placed on skin not covered by the blankets. These were also used in the cooling of the human patient; they provide some useful additional cooling, which can be readily removed when stabilization of the temperature is required.

The rate of cooling obtained with the human patient (about 4° C. per hour) is probably about as high as can be tolerated if satisfactory control of the subject's condition is to be maintained at all times.

A comparison of the results for the sheep clearly reveals the superiority of the blanket technique over that of cooling in a bath of ice-cold water from the point of view of the control the former provides for the stabilization of the subject's temperature. After sheep V was removed from the bath, a further drop of 2·5° C. occurred in the rectal temperature (in thirty minutes), whereas when blankets were used the overshoot, after cooling was stopped and the temperature had been allowed to drift to a halt, was only 1·1° C (sheep III). By deliberately raising the temperature of the water circulating in the blankets to well in excess of the final temperature desired (as in sheep II), only 0·3° C. overshoot occurred, and stabilization was obtained in ten minutes.

The rate of heat transfer to or from the blankets can be adjusted so readily by variations of water temperature that good control of the temperature of the subject at all times is solely a matter of the ability of the operator to judge from the temperature trend what adjustments must be made to produce the time-temperature schedule required. Under normal conditions control to about ± 0·2° C. should be easy to maintain.

The value of chlorpromazine (Dundee, Gray *et alii*, 1953; Ripstein *et alii*, 1954) for the control of shivering was confirmed in this work, and in addition it was noted that administration of this drug caused the rate of cooling of the subject to increase considerably, and greatly reduced the need for barbiturate even when no other anæsthetic or muscle relaxants were used. These effects were observed with both the sheep (for example, sheep III) and the human patient. It seems likely that the increased rate of cooling was due to peripheral vasodilatation (Foster *et alii*, 1954) rather than to the control of shivering, as it occurred when no shivering could be detected. Further support for this view is to be found in the difference between the rectal and peripheral temperatures. It will be

seen from Figure I that the curve for the temperature of one of the extremities of the sheep (just above the hoof) diverges from that for the rectal temperature well before shivering occurs, but is immediately brought close to it by the injection of more chlorpromazine. This suggests a possible method of controlling the administration of this drug so as to maintain its effect at a constant level.

## Conclusions.

A critical examination of the techniques which have been used for hypothermia leads to the conclusion that a method making use of a set of rubber blankets through channels in which water is circulated, supplemented during the cooling stage by the use of ice-bags, is the most attractive. Comparatively simple equipment constructed on the foregoing principles has been found to perform very satisfactorily. The particular advantages of the method are the following: (i) It is relatively easy to apply. (ii) The rate of change of temperature is as rapid as seems safe to use. (iii) There is full control of the subject's temperature at all stages. (iv) Cooling can continue in the preparatory stages of the operation, and warming can commence in its concluding stages. (v) Surgical access in an emergency is easy. (vi) There is no interference with asepsis.

Considerable emphasis has been placed throughout this paper on the desirability of good control of the subject's temperature. The problem of predicting the optimum degree of hypothermia which should be applied to any particular case is virtually unexplored. The use of methods such as that described above should greatly facilitate the obtaining of the data necessary to define the uses and limitations of hypothermia.

## Summary.

1. Methods of inducing hypothermia are reviewed and discussed.

2. A technique is described in which a set of so-called blankets is used.

3. Experiments on sheep in which this technique was used have confirmed that the method has a number of advantages over other methods.

4. The value of chlorpromazine in preventing shivering and assisting heat exchange by peripheral vasodilatation has been confirmed, and a possible method of monitoring its dosage is suggested.

5. The course of hypothermia is described for a patient on whom the foregoing techniques were used.

## Acknowledgements.

The development of the equipment and the performance of the work described here resulted from the cooperation and assistance of a large number of persons, not all of whom can be mentioned explicitly, but to whom the authors wish to express their thanks. Acknowledgements are particularly due to Dr. H. M. Windsor for his interest throughout the work and for his permission to quote particulars of his patient; to Dr. C. A. Cass for his cooperation at all stages and the operating theatre arrangements made by him at St. Vincent's Hospital; to Mr. J. Middlehurst and Mr. J. K. Braithwaite, of the Division of Physics, Commonwealth Scientific and Industrial Research Organization, for assistance throughout the work, particularly with the temperature measurements; to Dunlop Rubber, Australia, Limited, particularly Mr. Waring and Mr. Sharp, for the construction and donation of the blankets; to Mr. Ebsary, of Ebsray Pumps, Proprietary, Limited, for the loan of equipment used in the work; and to the Commonwealth Scientific and Industrial Research Organization, McMaster Animal Health Laboratory, for supplying two of the sheep and providing facilities for one of the tests.

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# Reviews.

**The Practice of Dynamic Psychiatry.** By Jules H. Masserman, M.D.; 1955. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 9¼" × 6", pp. 820. Price: £6.

THIS book reflects the "dynamic" personality of the author, whose experience concurrently lies in the experimental field of the animal laboratory and the clinical fields of hospital and private practice. He has developed a special philosophical approach to personality, and a practical approach to psychotherapy, based on an attempted synthesis of psychodynamic theory and the observation of experimental neuroses in animals. One feels that the bedfellows are not very compatible and that the marriage is of very uncertain outcome. Constant reference is made to the author's previous publication, "Principles of Dynamic Psychiatry", which must be regarded as a necessary companion volume to the present one.

These preliminary observations should not detract from the general interest of this book. It is vastly erudite and extremely stimulating. The more speculative, theoretical part of the book consists of some twenty pages, which, the author states, "may be omitted by those not immediately

concerned with its content". Although the rest is based on practical psychiatric problems, amply illustrated with 115 fully documented case histories, further theoretical dissertations and hypothetical meanderings abound throughout. The book begins with a section on psychiatric investigation, which aims to appraise the patient's "problems of adaptation" in relation to his previous development, present assets and current stresses. This is followed by a descriptive classification, using the United States "Standard Nomenclature of Nervous and Mental Diseases", but with emphasis on the behavioural tendencies shown and their possibilities of modification. The next section relates this approach to the presentation of reports to physicians, courts and insurance companies, as well as to job-placement; it seems to tend towards verbosity and an unnecessary complexity. The next section is the aforementioned philosophical treatise, which includes a discussion of concepts of emotion, personality, and cosmos, and their relation to an understanding of psychiatric phenomena. Lastly, over a third of the book is devoted to clinical treatment, ranging from individual psychotherapy to various methods of group therapy, and finishing up with an attempt to relate international social problems to problems of interpersonal behaviour. Appendices consist of an outline for psychiatric examination, the standard nomenclature of mental disorders, some data on alcoholism, the training of the psychiatrist, mental hygiene, and a bibliography of psychiatric literature covering forty-seven pages.

This volume cannot be regarded as a text-book of psychiatry for universal use. It reflects the dynamic pattern of change taking place in this field in the United States of America at present, with considerable emphasis on unverified psychodynamic theory, and inadequate consideration of basic biological science. Child psychiatry is almost entirely neglected and mental deficiency scantily disposed of in five pages. When one adds to this the author's own theoretical speculations, one can only conclude that it is a book which cannot be recommended to anyone other than the well-practised psychiatrist, who will be stimulated by its exuberant proliferation, will acknowledge the clinical practice, but will not necessarily accept much of the argument as more than the temporary and contemporary expression it reflects.

**The House Physician's Handbook.** By C. Allan Birch, M.D., F.R.C.P.; 1955. Edinburgh and London: E. and S. Livingstone, Limited. 7½" × 5", pp. 168. Price: 10s. 6d.

THIS handbook covers much the same general ground as others of its type and should be acceptable and useful to the young resident medical officer. There are chapters on general duties and information, clinical procedures, clinical pathology, treatment and the X-ray department. The description of general duties and information contains many small points that will greatly smooth the way and improve the work of the inexperienced young doctor starting his ward work; it includes such diverse information as a list of phrases in French, German and Italian that will be useful during the examination of foreigners, many medico-legal points, brief notes on embalming, and advice on certain religious matters, including the administration of baptism in an emergency. The most commendable feature of the book is the stress laid on the patient as a human being.

**Counseling in Medical Genetics.** By Sheldon C. Reed; 1955. Philadelphia and London: W. B. Saunders Company; Melbourne: W. Ramsay (Surgical), Limited. 8" × 5½", pp. 276. Price: 40s.

GENERAL PRACTITIONERS must be a little weary of being recommended treatises of portentous length and complexity by publishers. Dr. Sheldon C. Reed, director of the Dight Institute for Human Genetics, University of Minnesota, in his "Counseling in Medical Genetics" has presented to the medical profession a small, clear-cut and eminently practical booklet which will be most welcome. The young parents of an abnormal child do not consult an expert geneticist to find out if subsequent children are likely to be also abnormal; they go to the family doctor for advice; and if in breezy optimism they are assured that "lightning never strikes again at the same spot", they will lose confidence in his judgement if the lightning does strike at the same spot. Genetic information is also sought by relatives who would like to marry and also by childless couples who wish to adopt a child. Dr. Reed's opinion is that the medical adviser should not commit himself to a blunt decision that the young couple may or should not have children; his advice is that the perplexed inquirers should be given the probabilities in numerical form and be allowed to make their own choice. Patients are like medical students in that they appreciate simple, unambiguous statements, preferably expressed numerically, and do not find a spice of dogmatism unwelcome. Dr. Reed examines 24 of the most common abnormalities concerning which information is sought, and so with the aid of this book the probabilities can be given—certainty, 1 in 2, 1 in 4, 1 in 7, down to 1 in 20,000. The reader will learn that albinism is recessive whilst Huntington's chorea is strongly dominant, also how numerous "carriers" (heterozygotes) are in all communities. The chapter on racial admixture happily does not interest Australians. An appendix tells briefly about 128 additional diseases, whether they are dominant or recessive or are sex-linked. The occurrence of twins does not follow genetic law. Eye colour is useless as a guide to paternity. A warning given by Dr. Reed will gain the approval of Australian obstetricians—"The obstetrician should know whether his patient is Rhesus negative early in the pregnancy and if so whether the husband is Rhesus positive or not. If he is, proper precautions must be taken. It would seem that failure to do so should be adequate grounds for a malpractice suit." His opinion that "comprehension of the essentials of blood group genetics is a primary obligation of every physician" will also be applauded. Thanks to this admirable book the essentials of other branches of genetics can be readily acquired.

**Electrochemistry in Biology and Medicine.** Edited by Theodore Shedlovsky; sponsored by The Electrochemical Society, Incorporated, New York; 1955. New York: John Wiley and Sons, Incorporated; London: Chapman and Hall, Limited. 9" × 6", pp. 382, with many illustrations. Price: $10.50.

THE title of this book is somewhat misleading, since it is, in fact, an expanded version of a symposium with the same title, which was held in New York in 1953, under the auspices of the American Electrochemical Society. It is not by any means, as claimed by the dust jacket, "an authoritative survey of current work and thought in the field", since it is not in any sense a text-book on the subject, but rather a collection of papers by leading American specialists in each section.

The topics treated are the electrochemistry of semi-permeable membrane systems, bioelectric potentials, protein chemistry, molecular diffusion, sodium and potassium transport, electrocardiography and electroencephalography, a range which is scarcely likely to appeal in its entirety to any one reader. In general terms, the first three-quarters of the book is concerned with fairly adequate discussions, from several viewpoints, of current trends in electrophysiology, while the last quarter contains isolated topics, of some interest to the clinician, on the estimation of plasma proteins, the criteria for the selection of an electrocardiograph and of suitable electrocardiographic leads, and the application of electroencephalography to cerebral tumour diagnosis and to localization and to epilepsy.

**Neuro-Vascular Hila of Limb Muscles.** By James Couper Brash, M.C., M.A., M.D., D.Sc., LL.D., F.R.C.S.Ed., F.R.S.E.; 1955. An atlas with 30 coloured plates. Edinburgh and London: E. and S. Livingstone, Limited. 10" × 7½", pp. 100, with many illustrations. Price: 30s.

THIS is a monograph by James Couper Brash, Emeritus Professor of Anatomy, University of Edinburgh. The book is described as a small atlas, owing its origin to a suggestion made by Professor Sir James Learmonth in 1941 that accurate data on the sites and mode of entry of the principal arteries of supply to the limb muscles would be of great value in the diagnosis and treatment of the results of injuries affecting them.

The primary object was to determine the extent to which the principal blood supply of the muscles is associated with the nerves, that is, the constancy or otherwise of a definite "neurovascular hilum". This necessarily included checking and extending the observations already recorded on the points and mode of entry of the nerves of supply; and so, with the cordial concurrence of the Nerve Injuries Committee of the Medical Research Council, a detailed study on these lines was commenced.

It was obvious from the start that the collection of data from the dissecting room would take considerable time, and, in order to increase the observations on each muscle to a reasonable number, the collaboration of the departments of anatomy in the Universities of Aberdeen, Leeds and Glasgow (and at a later stage, in the case of the lower limb, the Edinburgh department) was sought and obtained.

The completion of the work, including special dissections from which the illustrations were drawn, and the radiography of limbs for the preparation of standard outlines, entailed much delay, and it was not until early in 1946 that it was possible to issue a typed brochure on the upper limb with illustrations printed from photographic stencils

and coloured by hand. Copies were distributed to all the principal nerve centres and to numerous individuals, both at home and abroad, but mainly in Canada and the United States of America.

The book consists of some thirty drawings of muscles and the associated nerves and blood vessels. Each nerve is taken separately, and a description of its mode of entry into the muscle is given. There is in addition a record of the very many observations made by numerous authors. In short, the book is one of great detail, and would be of value to all those whose work lies in the field of nerve injury or disease. It is a work which will repay close study and frequent reference to the various diagrams, which are very clear. The muscular and skeletal outlines are in black and white, while the nerves and blood vessels are shown in contrasting yellow and red.

At the end of the book there is a comparative table of the levels of nerve entry for both the upper and lower limbs.

**Statistics of Therapeutic Trials.** By G. Herdan, M.Sc., Ph.D., LL.D.; 1955. Amsterdam, Houston, London and New York: Elsevier Publishing Company; London: Cleaver-Hume Press, Limited. 9" × 6", pp. 384, with 80 illustrations. Price: 50s.

THE author maintains that there are five subdivisions of medical statistics — vital statistics, pathological statistics, medical genetics, biological standardization and therapeutic trials. One subdivision can be learnt without the others. It is evident, however, from the contents of the book that this arbitrary division breaks down.

It appears, moreover, that in any case the basic statistical theory and computations must be learnt, and it is immaterial from what field of study the examples are drawn. Certain general elementary texts have the advantages of clearer statement and of freedom from errors. In this book there is an inaccurate discussion of $\chi^2$. The treatment of Table V on page 47 is certainly not standard any more than is the Latin of *genius tempi, loci* and *epidemicus*. Nor can the book be regarded as a useful book of reference for the specialist. There is an extensive bibliography, not all of which seems relevant.

**Urology.** Edited by Meredith Campbell, M.S., M.D., F.A.C.S., with the collaboration of fifty-one contributing authorities; 1954. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. In three volumes. 10" × 6¾", pp. 2590, with 1148 illustrations. Price: £28 10s.

THE editor of this work, whose own private practice is largely confined to pædiatric urology, has had as collaborators able and distinguished surgical urologists practising in the United States, as well as physicians, pathologists and biochemical research workers. The only American contributor not from the United States is Luis Sanjurjo, of Puerto Rico. The only non-American contributor is Terence Millin, of London, who simply had to be invited to write a chapter on "Retropubic Prostatectomy".

In the section on "Anatomy and Physiology" in Volume I the anatomical descriptions are concise, practical and lucid.

A long section on "Principles of Diagnosis" by Gershom Thompson is followed by one on the "Pathology of Urinary Obstruction" by Frank Hinman, junior.

The next section, "Embryology and Anomalies of the Urogenital Tract", of 275 pages, is in the hands of the editor, Meredith Campbell. He states that "more than 10 per cent of all humans are born with some urogenital tract anomaly, and an anomalous organ is more prone to disease than a normal one".

In a section on "Infections and Inflammations of the Urinary Tract", Edward N. Cook deals with pyogenic infections, and Fletcher H. Colby with tuberculous infections.

Section 6 is a long hotch-potch section, devoted to "Infections and Inflammations of the Male Genital Tract". The diseases included range from ordinary diseases affecting the penile skin to syphilis.

Section 7, of 32 pages, is devoted to "Infertility in the Male". Discussion of the physiology of the testis, and of the complicated relationships between the hypothalamus, pituitary, adrenals and testis is clear and concise, yet it is admitted that in many respects our knowledge is as yet incomplete.

Section 8, by Charles Higgins, deals with "Urinary Lithiasis and Foreign Bodies"; only 70 pages are allotted to lithiasis, which seems inadequate.

Volume II commences with 90 pages on "Injuries of the Urogenital Tract". In the renal section A. J. School and P. A. Ferrier review 220 cases of renal injury seen at the Los Angeles General Hospital in sixteen years up to 1950. Most of the 44 deaths in the series were the result of coincident non-renal injuries. Injuries of the ureter are discussed by Carl Rusch and B. H. Hager. Injuries of the urethra, which deserve a chapter to themselves, are included in a chapter on "Injuries of the Genital Tract", in charge of James C. Sargent.

The next section is that of "Neoplasms of the Urogenital Tract". "Tumours of the Kidney" are considered by Clyde Deming. An enormous classification of tumours is given, and they are all discussed in the order of the classification. The result is that the reader becomes confused, and he cannot see the wood for the trees. From a practical point of view, nearly all renal tumours are malignant (as the author admits), and such practical points should be kept in mind even when constructing text-book classifications. Despite the present tendency to drop the term "hypernephroma", Deming keeps it and includes about 25% of adult renal tumours in this group, while he assigns a 34% incidence to adenocarcinoma. He calls the Wilms tumour a nephroma, since it contains all the elements of the kidney. "Tumours of the Ureter" are in the hands of W. W. Scott, who holds that the ætiology of primary carcinoma of the ureter is probably closely related to that of bladder tumours. Hugh Jewett writes on "Tumours of the Bladder". From the author's exhaustive studies it was found that the incidence of metastases was directly proportional to the depth to which the growth had invaded the muscular wall of the bladder. With only a half-way penetration, potential curability was 86%; with deeper invasion it sank to 26%. Two pathologists, R. V. Thomson and J. E. Ash, write on "Benign Hyperplasia of the Prostate Gland", the commonest disease treated by the urologist. The pathological lesion and theories as to ætiology, as well as the endocrine relationships of the disease, are generously discussed. Thomson and Ash favour the idea that there is an underlying endocrine disturbance of balance in favour of the œstrogenic hormones. William Wallace Scott, writing on "Carcinoma of the Prostate", states that the cure of this disease continues to be one of the major problems of urology. The actual cause, in spite of recent discoveries, is still unknown as regards the hormonal basis; no one, as yet, has observed production of prostatic carcinoma in man following prolonged androgen administration. Most theories fail to take into account the probable existence of a second hormone of the testis, œstrogen-like in nature, which would be reduced in amount following castration. In "Tumours of the Seminal Vesicles" Thomas E. Gibson states that primary neoplasms and cysts of the vesicles are extremely rare, and generally benign. When malignant tumours occur, they are nearly always secondary. The next chapter deals with a rather ill-assorted agglomeration called "Tumours of the Penis, Urethra, Scrotum and Testis". In discussing tumours of the testis, the author warns us that, though the seminoma is relatively benign, the clinician should recognize the deadly character of most testis tumours; seminoma constitutes only about one-third of the whole. The next chapter (by the same author) deals with tumours of the spermatic cord, epididymis and testicular tunics.

Section 10 is entitled "Neuromuscular Disease of the Urinary Tract". It contains several chapters.

Section 12, of over 40 pages, is entitled "Urology in the Female" and is in the charge of L. R. Wharton. He states that "backache of all sorts, and miseries in the lumbo-sacral region, seem to be the heritage of all women". The interpretation of difficult symptoms often calls for gynæcological, urological, orthopædic and neurological study. Pregnancy can be complicated by various renal malformations and anomalies, the most dangerous being a pelvic ectopic kidney. Adequate pelvic examination should reveal the ectopic kidney in many cases, excretion urography should show it in all. The incidence of uretero-vaginal fistula, even in the best clinics, is about 8%, and it occurs in major pelvic operations in spite of pre-operative ureteric catheterization. Wharton writes: "It is probable that in some instances the injury is not evident till years later, when an unexplained ureteric stricture, causing a large hydronephrosis, or a functionless kidney, is discovered."

The remainder of Volume II is taken up by a disproportionately large section entitled "Urology in Infancy and Childhood", written by the editor, Meredith Campbell.

Most of Volume III is devoted to descriptions of urological operations, but the first section of 30 pages deals with "Endocrinology in Urology".

"Surgery of the Kidney" is dealt with by James T. Priestly. In the surgical approach he is distinctly conservative. He does not use the "twelfth rib" or transcostal approach but sticks closely as his routine to the old classical subcostal incision. Resection of the twelfth rib is mentioned

only as an addendum during the subcostal operation, to facilitate access. The transperitoneal approach for large renal masses is well described.

In "Surgery of the Ureter", Thomas D. Moore wisely calls this duct a very delicate structure, and asks that its delicacy be respected in all surgical attacks on it. In discussing surgical approach to the lower third of the ureter, the most difficult portion to approach, he favours a low diagonal incision of the Gibson type. He only just mentions the mid-line extraperitoneal approach. He describes a special operation to which he has given the title of "protective ureterotomy"; this is a mere puncture with a small, spearpointed knife to allow passage of a ureteric catheter down through the lower part of the duct and on into the bladder. Moore wisely draws attention to the value of temporary ureterostomy to drain and rest a duct dilated by obstruction, the area of the latter being the site of operation; he uses a ureteric catheter for this purpose. In the following section, on "Plastic Surgery of the Ureter", the same author opposes the old idea of being resigned to nephrectomy when technically difficult plastic work on the ureter is demanded. He discusses several modern types of ureteric plastic operations. Finally, the very important subject of ureterosigmoidostomy is described.

The chapter on "Surgery of the Bladder" is by the editor. One must register an emphatic protest against an early editorial footnote which reads: "In everyday usage, rightly or wrongly, cystotomy and cystostomy are employed interchangeably"; the terms are not interchangeable. Good descriptions are given of types of diverticulectomy and of cystectomy for neoplasm.

The chapter on "Suprapubic Prostatectomy" is by Vincent J. O'Conor. It is a pity that, at this point in urological evolution, a good example was not set by this great work and the term "prostatectomy" abandoned except where the prostate is totally removed. For the usual enucleating operation in present cases, "adenectomy" may not be scientifically correct, but it is far less misleading. We must also take issue with the "studied opinion" of O'Conor that "every properly qualified urological surgeon should have the necessary skill and experience which would permit him to apply any one of the four methods of relieving prostatic obstruction". Perineal enucleation is not acceptable to most urologists simply because it entails the risk of serious morbidity. What is necessary is a mastery of two main methods—(i) either the transvesical (suprapubic) or the prevesical (retropubic) enucleation method, and (ii) endoscopic resection; the range of indications applicable to each of these two groups varies with individual urologists, but still must be available in each group.

J. Campbell Colston writes on "Perineal Prostatectomy".

The chapter on "Transurethral Prostatic Resection" is by Reed Nesbit. Nesbit states that the object of endoscopic resection is "complete ablation of the adenomatous tissue from the enveloping surgical capsule". The latter structure is the true prostate, thinned out. Therefore the operation achieves ideally exactly what is done by open "adenomatomy".

The chapter on retropubic prostatectomy is ably and clearly written by Terence Millin, of London.

Thomas E. Gibson writes on "The Surgery of the Seminal Vesicles", a subject much neglected even by urologists. It is strange that many urologists, though alive to the importance of the vesicles as the *fons et origo* of many distant infections and disabilities, do not pursue their diagnostic efforts so far as vesiculography; nor do they pursue their therapeutic efforts so far as vasotomy for vesicular medication, let alone prostato-vesiculectomy for intractable infections.

There is a section on "Radiation Treatment of Tumours of the Genito-Urinary Organs".

About 110 pages are devoted to the section on "Medical Diseases of the Kidney". These are of limited yet sometimes significant interest to the surgical urologist, but in these days he will enlist the aid of a consulting physician, or even transfer the patient entirely to him.

The final section of this gigantic work, "The Adrenals", is written by J. Hartwell Harrison and Dalton Jenkins. A practical description of the surgical anatomy of these glands is given. In a description of adrenal physiology the authors state that during the past two decades 28 different steroid compounds have been isolated from adrenal cortical extracts. Adrenal cortical insufficiency is considered in both its chronic and its acute manifestations, and then adrenal cortical hyperfunction and tumours of the adrenal cortex. The concluding discussions are on the physiology of the adrenal medulla and its tumours, and on adrenal surgery in systemic diseases, such as carcinoma of the prostate and of the breast.

## Notes on Books, Current Journals and New Appliances.

**Family Doctor.** Published monthly by the proprietors, the British Medical Association, Tavistock Square, London, E.C.1. Sole agents for Australia and New Zealand: Gordon and Gotch (Australasia), Limited. Subscription for twelve months: 20s. (sterling), including postage.

WITH a charming cover and a special Christmas supplement covering such things as festive cooking and Christmas presents, the December issue of *Family Doctor* is as attractive as ever. Special articles deal with the problems of a stepmother and how they were met, the giving of presents by children, osteoarthritis, the medical detective work involved in investigating an outbreak of trichinosis, children and belief in Father Christmas and fairies, Mrs. Beeton (of cookery book fame), the nails, alcohol and other subjects. All the regular features are present, making up a first-class issue.

## Books Received.

*[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]*

"Cancer Cells", by E. V. Cowdry; 1955. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 10" × 6½", pp. 694, with 137 illustrations. Price: £8.

The author is director of Wernse Cancer Research Laboratory, Washington University, St. Louis.

"The Year Book of General Surgery (1955-1956 Year Book Series)", edited by Evarts A. Graham, A.B., M.D.; 1955. Chicago: The Year Book Publishers, Incorporated. 7½" × 5", pp. 656, with 182 illustrations. Price: $6.00.

One of the Practical Medicine Series of Year Books.

"Textbook of Endocrinology", edited by Robert H. Williams, M.D., with ten contributors; Second Edition; 1955. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 9½" × 6", pp. 788, with 173 illustrations. Price: £6 10s.

The first edition was published in 1950.

"Minor Surgery", by John E. Sutton, M.D., F.A.C.S.; 1955. New York: Landsberger Medical Books, Incorporated. Distributed by The Blakiston Division of the McGraw-Hill Book Company. 8" × 5½", pp. 334, with 102 illustrations.

This is one of a series of "Handbooks for the General Practitioner".

"Adaptive Human Fertility", by Paul S. Henshaw, Ph.D.; 1955. New York, Toronto, London: The Blakiston Division, McGraw-Hill Book Company, Incorporated. 9" × 6", pp. 336, with 28 illustrations. Price: $5.50.

The author attempts to deal with human fertility and population growth.

"Group Processes: Transactions of the First Conference, September 26, 27, 28, 29 and 30, 1954, Ithaca, New York", edited by Bertram Schaffner, M.D.; 1955. New York: The Josiah Macy, Jr. Foundation. 9" × 6", pp. 334, with 40 illustrations. Price: $5.50.

The subjects dealt with are: "Ontogeny and Living Systems", "Psychology and Ethology as Supplementary Parts of a Science of Behavior", "Morphology and Behavior Patterns in Closely Allied Species", "Dynamics of the Mother-Newborn Relationship in Goats", "Perception of Animal Behavior", "Group Processes in the Lower Vertebrates".

"Acta Leidensia" Edita Cura et Sumptibus, Scholæ Medicinæ Tropicæ; 1955; Volume XXV. Leiden (Holland): Universitaire Pers Leiden. 9½" × 6½", pp. 236, with 23 illustrations.

Deals with the host-seeking behaviour of the malaria mosquito and with alastrim.

The Medical Journal of Australia

SATURDAY, JANUARY 28, 1956.

*All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words of phrases.*

*References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.*

*Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.*

## INFECTIOUS DISEASE: A CONTINUING PROBLEM.

THE present status of infectious disease in the health of the community demands careful thought. In the course of his Listerian Oration delivered in April, 1952, Sir Macfarlane Burnet[1] said: "In 1952 we can look upon infectious disease as conquered." In its context this statement is not so bald as it appears here; there are qualifying statements, such as: "We can never feel quite confident that there are no unpleasant surprises in store for us amongst the virus diseases." Nevertheless, the sentence was apparently intended to convey in general terms just what it stated; and Burnet was speaking with authority. He was satisfied that for the great majority of infectious diseases, measures of control and treatment had reached a high level of effectiveness; and, apart from a somewhat increased uneasiness about the effect of indiscriminate use of antibiotics, nothing much has happened since the statement was made to invalidate it. We may fairly be optimistic about the control of infectious disease. At the same time, we may not be blindly optimistic, and certainly we may not relax our guard. The conquest may be complete in the eyes of the generals, who think (quite rightly) in terms of grand strategy; but in the field the battle goes on against a stubborn enemy, who is disconcertingly ready to change his tactics and counterattack in unexpected places. In the first of four papers read at a plenary session on infectious disease at the last congress and published in this issue, H. McLorinan affirms the fact that infectious diseases still comprise an important section of medicine. He states that general practitioners to whom he has spoken estimate that 75% of the medical illnesses with which they are concerned are infective in origin, and he goes on to quote authoritative statements from the United Kingdom and from the United States of America showing the continuing major role of infectious diseases in the production of sickness and death in both places.

[1] M. J. AUSTRALIA, June 14, 1952.

The position in Australia from the public health viewpoint is admirably summarized by D. W. Johnson, who, while by no means pessimistic, offers no grounds for complacency. Clifford Jungfer, presenting the general practitioner's experience, is equally realistic. It is to be hoped that these papers and that by J. A. R. Miles on diseases of animals transmissible to man will be carefully read, and that something will be done about the constructive suggestions, especially those of McLorinan and Jungfer, which are designed to promote valuable cooperation between general practitioners, hospitals and public health authorities.

Meantime we should like to refer to a comprehensive survey of the position of infectious disease in the United States presented recently by Theodore J. Bauer,[1] Chief of the Communicable Diseases Center, Public Health Service, United States Department of Health, Education and Welfare. He sets his readers thinking with an opening statement that communicable diseases take "an immense toll in death and disability among the citizens of the United States". Of every ten deaths that occur, one is caused by a communicable disease. In the older age groups the ratio is 1:12, but amongst those under thirty-five years of age it reaches the more formidable figures of 1:4. In addition, there is a considerable morbidity rate. About a million and a half cases of infectious disease are reported annually, and to these must be added cases of non-notifiable disease, including pneumonia, influenza and other upper respiratory tract infections, various gastro-intestinal infections, and many less defined infectious states. Bauer states that the infectious diseases, as a whole, account for the majority of absences among the working population and among school children, and they are the main cause of absences from duty in the armed forces. Another increasingly important source of loss is in the various sequelæ in the middle and late years of life resulting from infectious diseases contracted during childhood and youth—rheumatic and syphilitic heart disease, chronic nephritis of streptococcal origin, chronic fibrosis of the liver from viral hepatitis, and so on. An estimate of losses due to infectious diseases in terms of dollars is difficult to make, but Bauer states that the total is in billions and points out that sales of antibiotics alone amount to approximately one billion dollars annually—this refers, presumably, to the American billion of one thousand million. He then goes on to refer to the fact that within the present century some of the most spectacular and dreadful of the infectious diseases, among them yellow fever, typhus, plague and smallpox, have been reduced to such an extent that they are considered to be of minor importance. However, the degree to which any one of them is held in check depends on the extent to which time-tested as well as modern control measures, including the more recently discovered techniques of chemotherapy, are applied. Bauer writes:

> The infrequency with which such diseases are seen does not mean that they have been eliminated as matters of public concern; rather, it is an indication that death and illness from the major killers of half a century ago can now be largely prevented through immunizations, vaccinations, proper sewage disposal, protection of food and water supplies, destruction of disease-bearing insects and rodents, prompt attention to focal points of infection in the environment and the population, quarantine activities, and by the high levels of personal hygiene that have become a part of our way of life.

Bauer then goes on to consider individual infectious

[1] *J.A.M.A.*, August 20, 1955.

diseases and the problems they raise; but it is not possible to follow him into a detailed discussion. It may be sufficient to point out that virus diseases still constitute a major problem, as virology is a very young science, knowledge of the viruses is still fragmentary, and measures of both prevention and treatment are in many cases far from satisfactory. Among the bacterial diseases, on the other hand, the therapeutic triumphs of the antibiotics have been spectacular—only to lose a good deal of their lustre as the emergence of resistant strains has become apparent. Bauer points out that control of these strains of bacteria requires extended knowledge concerning the fundamental nature of the mechanisms involved in the mode of action of the antibiotics and of the factors involved in the development of resistant strains; equally important is further clarification of the epidemiology of infection by the resistant and the non-resistant strains.

The most cursory glance at the history of disease shows changes in the nature of individual diseases that are not fully understood. A classical example is the variation in the virulence of syphilis from the acute terrifying contagion of the fifteenth century in Europe to the extreme tolerance reported in 1916 by H. C. Brown (and quoted recently by E. G. D. Murray[1]), who found that nearly everyone in a village in the Punjab gave a positive Wassermann reaction, although no stigmata could be found. Scarlet fever was a dreaded killer, especially of children, during last century; today for no very obvious reason it is almost a minor ailment, although many share the view of D. W. Johnson that "a virulent phase will recur". There is widespread doubt that the decline in the incidence of diphtheria is wholly due to immunization measures, even while the value of such measures is not questioned. And so we might go on. There has been a constant ebb and flow in the seriousness of infectious diseases. Beyond doubt the changes have involved both the susceptibility of the host and the virulence and nature of the organism. Variations in host susceptibility are in their turn complex; they are governed by much more than the presence of specific acquired immunity, according to E. G. D. Murray, to whose long but fascinating paper on "Destiny and Determinism in Infectious Disease" we have already referred; perhaps the old conception of diathesis is awaiting restatement in modern terms. Variations in organisms have occurred with disconcerting frequency and apparent facility in the face of changing conditions and arouse considerable speculation in relation to the challenge of antibiotics. Bacteria would seem unwilling to be relegated to museums or to see their masters, the bacteriologists, lowered to the status of "minor members of the Department of Archæology" (the phrase is Murray's).

Readers of this journal are probably weary of constantly repeated warnings about the dangers of the indiscriminate use of antibiotics; but we must plead that the issues involved are of far-reaching importance. The immediate dangers to the individual patient are real enough, but at least they tend to be clinically apparent and so impress the practitioner. The subtler and more dangerous effect is the change in the nature of pathogenic organisms. Their adaptability in response to the demands of natural selection has been amply demonstrated in the past; they can be expected to respond wholeheartedly to the challenge of modern antibiotic therapy. In a recent review of the problems of antimicrobial therapy for infectious diseases Ernest Jawetz[1] quotes a statement by Lord Horder that ". . . although many epidemic diseases of previous centuries have disappeared the automobile has almost replaced the microbe as a menace . . ."; but Jawetz's review attempts to point out that although the vast developments in antimicrobial therapy serve as powerful tools for the physician in his attempts to control infection, they have not removed "the microbe as a menace". The Hunterian Society in London debated, on November 17, 1952, the proposition "that the continued advance in medicine will produce more problems than it solves" and, after several somewhat facetious speeches, carried the proposition by 59 votes to 47. Jawetz remarks (somewhat drily, we presume) that the adjustment of the microbial world to the impact of chemotherapy serves as an illustration that the majority opinion may be correct. This is surely too pessimistic a view. If we were prepared to spend on medical research even a fraction of what we spend on agents of potential destruction, and if we were all prepared to approach clinical practice in a scientific spirit, we should be able to realize in actual fact the conquest of infectious diseases which to men of vision like Burnet is already a reality.

[1] *Canad. M. A. J.*, May 1, 1955.

# Current Comment.

## THE MATHEMATICAL THEORY OF EPIDEMICS.

THE reading of a paper on the mathematical theory of epidemics followed by a discussion at the Royal Statistical Society (by N. T. J. Bailey,[3] 1955) and two reviews, one by H. Abbey[3] and the other by R. E. Serfling,[4] enable us to examine the progress that has been made in this field.

Sir Ronald Ross, a highly competent mathematician, devoted much of the later part of his life to the consideration of epidemics by mathematical means. He used what are now known as deterministic models, that is, the play of chance was excluded. For example, in an epidemic if 100 persons were each exposed to a 10% risk of infection, 10 would be imagined to be infected. As in the application of mathematics in other fields, the general procedure is to abstract from observations the salient features of an epidemic such as infectiousness. This will be defined in the model as the probability of one member infecting a second, and allowance will be made for such variable factors as the relation of one person to another, whether they belong to the same group such as the family, school or other social unit. The latent period before a person becomes infective to others can be defined and used in the model and so can other relevant epidemiological concepts. Appropriate numerical or algebraic values are given wherever possible. Ross used such methods to obtain the form of the epidemic curve.

Useful results were obtained from these deterministic models and the mathematics were relatively simple. However, some epidemiologists, notably A. G. McKendrick, felt that a more realistic treatment was only possible if the chance element was introduced. The model is then said to be stochastic (Greek στόχος, aim or guess). The theory of such stochastic models has made great progress in recent years because of its interest in nuclear physics. Bailey has done much in applying the stochastic theory to epidemiology.

[1] "Annual Review of Medicine", 5:1.
[3] *J. Royal Statist. Soc.*, Series B, 1955, 17, 35.
[3] *Human Biology*, 1952, 24, 201.
[4] *Ibidem*, 1952, 24, 145.

The stochastic theory shows that, given any initial conditions, a great variety of epidemic forms can result. But in general, some forms of epidemic wave are much more probable than others and these usually resemble those derived by the deterministic theory. This type of analysis shows that many apparent changes in epidemic behaviour during an epidemic can be explained by chance effects. Field epidemiologists have, perhaps, called upon more concepts, such as enhanced virulence during the epidemic wave, than might be necessary. From these mathematical studies improved methods of estimating infectiousness, length of the latent period and other numerical features of infective disease are emerging. The stochastic theory also shows the impossibility of forecasting the future of an epidemic, although the most probable types of outcome can be given. A notable feature of the discussion on Bailey's paper was the large number of medical workers in London taking part. Of past workers, mention was made of Farr, Brownlee, Ross, McKendrick and Greenwood.

## THE EAR AND THE CAISSON WORKER.

THE problem of noise in industry continues to increase as mechanization becomes more general. The widespread deafness found in the past in those who tended boilers, minded cotton looms or guided high-speed vibrating drills has usually been controlled by the improvements in working conditions and by the perfecting of mechanical efficiency, which are often accompanied by a fall in noise level. However, as the specific occupational hazards disappear, more generalized noises arise in industry; so that the psychological and physical problems arising from high noise level, while no longer acute, are increasingly widespread. Occupational diseases of the ear result not only from noise, but also from profound changes in atmospheric pressure. The trauma which results arises rapidly, causes severe permanent damage to the whole of the sensory organ of the eighth cranial nerve, and is preventible. Those in danger owing to uncompensated rapid changes of atmospheric tension are high-altitude flyers in unpressurized aircraft, deep-sea divers and caisson workers.

Caisson workers are employed for projects involving tunnelling under water or through soft deep soils. Their working conditions have been described by D. Hunter.[1] As the caisson gradually sinks to the desired depth, compressed air is used in order to overcome the pressure upon the sealed chamber of the material which surrounds it. Workers enter and leave the caisson through a series of air locks or compression chambers, and they leave after the working shift in the usual way. Severe trauma is less likely to occur in flyers and deep-sea divers, as these are exposed to atmospheric changes for only a short time and at less frequent intervals. Caissons are usually worked at a pressure of up to 35 pounds per square inch, but in one hour shifts atmospheric pressures of up to 50 pounds per square inch have been used. In addition to the high atmospheric tension there is usually a high noise level. Caisson workers are highly paid, have a short working day and tend to remain in this kind of employment for a large part of their working life.

A group of caisson workers was recently studied by J. C. Seal,[2] who discusses the ætiology of the signs and symptoms of decompression trauma. Several of the workers claimed that they were now deaf as a result of their occupation. They had all been working in compression chambers for over twenty years, and none admitted more marked symptoms than occasional slight blockage of the ears on entering the chamber; a few had noticed occasional epistaxis. Clinically most of them were found to have dull, retracted, thickened and sclerotic tympanic membranes. There were several with old healed perforations, and in most of the ears the tympanic periphery was calcified and no light reflex could be obtained. In some cases the ossicular chain was surrounded by chalky sclerosis, and the X-ray findings were of advanced sclerosis of the mastoids. Audiometer readings demonstrated a general loss of both low and high tones, with the usual nerve deafness depression in the hearing acuity at a 4000 cycles per second sound frequency.

During compression, when the atmospheric pressure rises, blockage of the Eustachian tube results in retraction of the drum and collection of fluid in the middle ear. The condition is usually rapidly relieved by a Valsalva technique. Decompression results in the opposite and more damaging effect with the building up of pressure in the middle ear and the possible bursting of the drum. Symptoms of over-rapid uncompensated decompression are a sensation of pressure in the ears with deafness and pain. Deafness may be transitory when relieved by Eustachian ventilation. In more severe phases deafness may last for from four to fourteen days, and the drum is found to be congested with possible signs of hæmorrhage into the drum or middle ear and with possible bleeding from the Eustachian tube into the sputum. Recovery of auditory function is not usually complete. In the most acute cases there are vertigo, nausea and vomiting, with all the signs of a Ménière's syndrome and almost certainly some permanent deafness. The "bends" familiar to divers occur usually before the auditory signs and are characterized by pain in the extremities and the abdomen and by neurological signs. The "chokes" may also occur with upper respiratory symptoms of acute tracheitis or bronchitis with mucous congestion and dyspnœa. However, acute auditory damage does often occur as an isolated event. Deafness usually results if hæmorrhage takes place into the cochlea and semicircular canals. After perforation of the drum there is usually a hearing loss of 25 to 35 decibels with whole-range diminution of acuity. In those patients whose deafness is temporary, acuity loss is more apparent over the low-frequency range. When the most acute symptoms occur, the condition is due to the release of nitrogen and carbon dioxide bubbles causing emboli in the middle ear and labyrinth with permanent deafness and Ménière's syndrome. Severe migrainous headache may accompany the symptoms and is due to cerebro-vascular bubble formation and spasm. During the early acute phases recompression followed by gradual decompression may reverse the process.

The methods of preventing these traumata are obvious. Changes in atmospheric pressure must be brought about gradually, and the workers must be carefully supervised to ensure that this takes place. They should be familiar with the simple methods of opening the Eustachian tube should blockage occur. Finally no worker should be admitted to the caisson in whom there are signs of tonsillar infection, nasal polypi, hypertrophied lymphoid tissue in the naso-pharynx, dental abnormalities or ordinary infections of the throat.

[1] "The Diseases of Occupations", English University Press, London, 1955.
[2] *New York J. Med.*, October, 1955.

## ACCLIMATIZATION TO HIGH TEMPERATURES.

OWING largely to changes in peripheral dilatation of blood vessels and sweat secretion, man is able to maintain a relatively constant temperature in the face of very large changes in environmental temperatures. It is a matter of common observation, however, that with the sudden advent of hot weather there is an impaired ability to do work which could easily be done in the cooler temperatures. After a few days of exposure to the higher temperatures the ability to work largely returns. On a larger scale similar changes are seen in passing from a temperate to a tropical climate. The process of change is termed acclimatization. If one lives in a tropical climate for some time the ability to work increases still further and the feeling of discomfort engendered by the heat becomes much less. Studies on acclimatization, both the short-term process and the long-term change, have been extensive over the past forty years, but it cannot be said that the changes in the physiological processes which account

for the acclimatization are at all well understood. The earlier work was done on people living in the tropics, but here the work was very difficult because it was almost impossible to get adequate controls. For many years most of the observations have been made on individuals living in a temperate climate and exposed to high temperatures in hot rooms for relatively short periods. Observations of this type have been carried out in considerable detail by a group of workers at Natick, Massachusetts, and Boston University. D. E. Bass, C. R. Kleeman, M. Quinn, A. Henschel and A. H. Hegnauer have published the results.[1]

In a review of previous work the authors state that it has been found that the improved ability to work in the heat is accompanied by certain well-defined physiological adaptations. These are: (*a*) lower skin and rectal temperatures during work; (*b*) lower heart rate during work; (*c*) increased cardio-vascular stability with changing posture; (*d*) increased sweat rate during work; and (*e*) decreased metabolic cost for a given task (that is, greater efficiency). Attempts have been made to explain these changes in terms of altered pituitary-adrenal activity. The authors have studied five healthy young soldiers acclimatized to heat by living continuously for fourteen days in a constant temperature chamber maintained at 120° F. for twelve day-time hours and at 100° F. for twelve night hours. Very extensive observations were made, including determination of total body water, plasma volume, adrenal cortical activity from measurements of circulating eosinophile cells, metabolic balances for nitrogen, phosphorus, potassium, sodium, chloride and water. In addition hæmoglobin level and plasma protein content were measured, the urine was examined for many constituents and so on. Pulse rates and rectal temperatures were determined frequently. As a result of these observations the authors conclude that the most important adaptations are those concerned with cardio-vascular function, thus confirming the claims of several previous observers. The improved body temperature was the result of blood volume changes which permitted a more rapid transfer of heat from the body core to the surface. The more dilute character of the sweat reduces solute losses for a given sweat rate and permits readier evaporation of sweat water. The reduced efficiency of working during the earlier exposure to heat is probably the result of marked enlargement of the vascular bed due to cutaneous vasodilatation without immediate increase in blood volume. With exercise the demand by the muscles for increased blood flow results in further discrepancy between the size of the vascular bed and the blood volume. There result then signs of peripheral vascular collapse such as dizziness and very rapid pulse rate during work. As blood and interstitial volume expand, as they do in the first few days of acclimatization, the cardio-vascular responses improve. After maximal expansion of the extracellular fluid has occurred, no appreciable further improvement took place. During the process of acclimatization there is a renal retention of sodium and chloride in excess of that required to make up for the increased loss in the sweat. Nitrogen, phosphorus and potassium balances were negative during the heat period and positive during recovery. There was no evidence that changes in the pituitary-adrenal system played any important part in acclimatization. "Although one speaks of 'acclimatization to heat', it would appear that the early dramatic improvement in ability to work in the heat is related more to protection against circulatory disturbance than against elevated body temperature." The detail and thoroughness of the work in these observations make it probable that the explanation given by the authors for short-time acclimatization to heat is correct, but it is equally obvious that the explanation is not adequate, or at any rate not complete, for acclimatization to a tropical climate. This acclimatization takes a relatively long time and is more complete, and there is also what one may call a negative acclimatization when one returns from the tropics to a temperate climate. What may appear to be a cool day to the resident of North Queensland may be uncomfortably hot to a visitor from the south, and on the other hand a visitor from North Queensland to the south will feel a day which is comfortably warm to a Sydney dweller as decidedly cold, and it takes a considerable time for this feeling of increased cold to be lost. Furthermore it is obvious that the negative balance of certain substances cannot persist in a dweller in the tropics.

[1] *Medicine*, September, 1955.

## NUTRITION AND THE AGING.

ONE of the future problems of civilization will lie in the increasing dominance of the older age groups. The conception that death relieves society of those too old to work may still be true. But the ability of man to contribute the labour of his skills and the products of his wisdom increases at the end of his life. Man lives longer and dies when he is no longer of use. The rigid demarcation by age of the useful from the useless is unrealistic and very wasteful. Retiring age cruelly cuts out the usefully mature, thrusts them out into cloying penury and saps at their determination to survive. In an aging society there is no room for the prematurely idle. Individual fitness of mind and body must be maintained for as long as possible, otherwise old age becomes a mere achievement without reason. The problems of aging are many and are to some extent artificial. It is apparent that mental deterioration with advancing years is due often not to impairment of the intellect but to loss of the will to use it. The body withers as its eagerness to thrust forward in the race of competition is lost. Old people lose by default their powers of adaptation. In an attempt to linger on in the memories of youth they become detached and lonely. In losing husband or wife they lose their hopeful love, in losing hope they fall into self-neglect. Young families grow out into their own lives and either selfishly abandon their parents or well meaningly wrap them into over-attentiveness. Far from restricting the mental and physical activities of the old, they should be encouraged to continue and to foster their interests. They must live and know that they live as active and useful units in the family or group. However, the body as a physical whole may be showing the signs of wear and tear. The rate of metabolism is decreased, reparative processes are slow, exercise tolerance is progressively limited, and the efficiency of the musculature, the circulation and the gastro-intestinal system is gradually reduced. F. J. Stare[1] has studied the particular problems of nutrition in the aged person. He believes that a well-balanced diet is just as important for the old person as for the child or young adult. Physical activity is essential for the enjoyment of healthy appetite, the elimination of waste and the prevention of immobilizing and vitality-sapping obesity. In the reduction of weight by diet, sodium and water retention must be avoided as otherwise the patient is disappointed at the apparent failure to lose weight. Obesity and hypertension are the commonest physical defects in those over fifty years of age. Surveys conducted by the Harvard School of Public Health have shown that old people tend to eat more than their Calorie requirement and that about half of them are 10% or more overweight. Muscle is replaced by adipose tissue and life insurance statistics demonstrate the adverse effect of obesity upon longevity. Recent work has suggested that proteins from different sources are most efficiently utilized when taken together. Nitrogen equilibrium is sometimes not maintained and there may be a distaste for protein foods. In these cases artificial substitutes are indicated. However, proteins and their substitutes are expensive. Stare suggests the provision of a cheap cereal food such as bread, enriched by the addition of amino acids such as lysine. Defective protein is often related to fatigue, œdema, anæmia, lowered resistance, chronic exematous dermatosis, senile pruritus and bed sores. Correction of the hypoproteinæmia often brings about useful improvement. Studies in atherosclerosis in the monkey indicate the advisability of reducing fat intake with the advancing years. Atherosclerosis is produced by an elevated and

[1] *Am. Geriat. Soc.*, October, 1955.

maintained serum lipid level. The pathological process is produced by a high dietary fat and cholesterol level with, at the same time, a deficiency in the sulpha amino acids, methionine and cystine. Choline must be present in sufficient amounts for the atherosclerosis to take place and the methyl group of methionine is probably not involved. The protein component concerned in the process is the organic sulphur. The level of blood lipids certainly tends to be high in the obese and to be lowered with a reduction in weight, but this finding is not universal. Sudden gains in weight in the normal individual are usually accompanied by a rise in serum lipids. The role of the phospholipids in the production of atherosclerosis is as yet uncertain. In the presence of good health and with an adequate diet vitamin supplements are unnecessary. The problems of aging are intimately bound up with the maintenance of healthy physical and mental activity. Avoiding an excess of carbohydrate Calories and a predominance of protein intake over fat not only would promote the healthful longevity of the aged, but might well improve the vigorous health of the young and delay the ravages of the advancing years.

## PROTOPHRENIA.

THE awakening of the social conscience has done much towards dispelling the more obvious forms of individual injustice. Physical deprivation and apparent child cruelty are gradually disappearing from Western society, and the readjustment and social rehabilitation of the mildly maladjusted are progressively facilitated. But society is still bedevilled by the aggressive behaviour of a small group of its younger members, and the causes which lead to diversions from the accepted code of behaviour are still largely obscure. Much study is needed to seek and to eliminate any cause which denies happiness to the individual and security to society. The influence of extraneous factors in the causation of the huge depressing numbers of the mentally defective has not until recently been considered worthy of research. But now it seems that responsibility for a considerable proportion of these hopeless human derelicts lies within society itself.

In a recent study H. Bourne[1] investigated the behaviour patterns and social background of 154 mentally defective young children admitted over a period of two years to the Fountain Hospital, London. The object of the survey was to assay the influence of psychological adversity in infancy, and to discover the prevalence and features of any resulting psychogenic amentia. It had been noted in the past that there was always a group of mental defectives in whom there was no apparent organic cause for the abnormality, who showed a curious pattern of abnormal behaviour and who had a background of severe social misfortune. It is already known that severe emotional trauma in the young may disrupt basic mental development. It is also known that the establishment of animal behaviour patterns depends upon stimuli normally encountered in the particular species. Thus abnormal stimuli result in abnormal behaviour patterns and, likewise, we may conclude that in man mental dwarfism may follow distorted infantile experience.

The children used in this survey were not selected. They were first divided into two groups—those with and those without an organic cause for their mental condition. All the children investigated had an intelligence quotient of less than 50. Of the children, 138 had an organic cause for their mental condition and 16 had none. The organic causes are specified in the article. Investigation was made into the family background, the pregnancy and the labour, use being made of medical records, interviews with parents and relatives, social agencies and local authority workers. The findings were tabulated and compared statistically.

There were more males in both groups. Intelligence was measured on the Vineland scale. The intelligence quotient of the residual groups was found to be higher on the average than that of the organic groups, but a subgrouping of children of similar intelligence quotient from the organic group gave the same results as the whole series. As expected, the mean maternal age in the mongoloid children was higher than that of the others in the organic group, but that of the residual group was also the same as the majority when the mongols were eliminated.

The incidence of pregnancy difficulties was the same in each group, and once the mongoloid subgroup was again eliminated there was no significance to be attached to difficulties in labour. There was no difference in the incidence of familial mental defect. There was no significant difference in the proportion of twin births, but it was noted that the single twin in the residual group had apparently a normal sib who was also reared differently. There was no difference between the two groups in the size or the economic status of the families. However, when the incidence of neonatal abnormalities in the organic group was 49%, it was four times as high as in the residual group.

The role of the father was not found to be of great significance when the emotional background of these children was investigated. Defects in the mother were taken as either qualitative, where there was maternal mental abnormality, or quantitative, when there had been prolonged absence from the mother's care. It was here that very significant differences occurred between the two groups. The residual groups had high rates of all types of pathological mothering. Illegitimacy in itself was not significant, but discordant marriages before birth were seven times as common in the residual group as in the organic group, and the incidence rose from 28·6% to 42·9% in the neonatal period. A higher percentage of siblings were also abnormal among children of the non-organic residual group, and there was a considerably higher tendency towards antisocial behaviour in relatives.

Abnormality in the mental development of the child was always noticed first in the organic group. This may be because many infantile abilities such as sphincter control, not being so greatly influenced by psychological factors, would develop at first normally in children of the residual group. Thus the picture of the psychogenic mental defect emerges — a child physically healthy and mentally backward from the second year of life, with behaviour abnormalities and a history of disturbed mothering, in that either the mother is a psychopath or the child is denied the mother's care. The children outwardly do not appear to be mentally defective, and they are either very detached or very antisocial. There are about 10% of all mental defectives in this residual group. They may be said to be suffering from "protophrenia", that is, a psychogenic failure of ego formation. This is probably the second most common cause of severe amentia.

The survey by Bourne is obviously too small in numbers to be statistically conclusive, but the indications are strongly in favour of a purely infantile psychosis with a resultant amentia. Further work is needed to find out when, if at all, it is possible to reverse the process of amentia. Also this survey throws more light on the mentally defective child who has alternated in his infancy between his neglectful unhealthy home and hospital. The withdrawal from an already inadequate mother may perhaps accentuate the child's psychosis, and the frequent visiting of the infant patient in hospital may be even more important than previously thought. A poor mother may be better than none at all, or the infant must be provided with an alternative stable mother figure as soon as possible. It is interesting to note that the picture in these children is similar to that of an acute schizoid state. Perhaps in reacting as they do, even under severe provocation, these children are merely demonstrating some basic mental inadequacy. Further, one may argue that the unstable child is the heir to the instabilities of the parents, so natural selection may result in some eradication of the inherited character. But in many of these children there is no parental abnormality and the children are the victims of social misfortune. Can there be any greater tragedy than that at the dawn of its consciousness a human being may find a world so hostile that it can reject its own humanity?

[1] *Lancet*, December 3, 1955.

# Abstracts from Medical Literature.

## PATHOLOGY.

### Pancreatic Islet-Cell Adenomata.

H. SPENCER (*J. Path. & Bact.*, January-April, 1955) discusses a series of 19 cases comprising 28 islet-cell adenomata; five cases were associated with hypoglycæmia, two with hyperglycæmia and the remainder with no clinical evidence of either. The author states that pancreatic islet-cell adenomata are more common than is at present generally realized, but thorough examination of the pancreas is essential if they are to be discovered. The insulin-secreting tumours are encountered mainly in patients under the age of fifty years, the non-secreting in persons above this age; the difference is statistically significant. A case of generalized islet-tissue hyperplasia associated with multiple adenomata enabled the origin and growth of these tumours to be observed at all stages. Islet tissue was seen to arise from small pancreatic ductules. The non-insulin-secreting tumours were frequently found in pancreatic glands which showed wide-spread evidence of ischæmic change. Two adenomata, including one arising from ectopic pancreatic tissue in the wall of the duodenum, displayed an acinar structure relating them more closely to ductular or even pancreatic acinar tissue. The whole problem of secretory and non-secretory pancreatic islet-cell tumours has been considered in relation to similar tumours arising in other endocrine glands.

### Hæmorrhage, Necrosis and Cyst Formation in the Thyroid Gland.

N. JOHNSON (*Surg., Gynec. & Obst.*, July, 1955) describes a series of eight cases of hæmorrhage, necrosis and cyst formation in thyroid nodules and reviews the literature pertaining to them. He discusses the significant association of the presence of giant vessels or of dilated tortuous vessels, occurring in areas of epithelial activity, and necrosis in thyroid nodules. He suggests that the following sequence of events will explain the degenerative changes seen within such thyroid nodules. Vascular disturbances resulting in ischæmia may be due to the following causes: (i) mechanical obstruction of the nodular artery or one of its branches (that is, thrombosis in the vessel); (ii) functional disturbances, which may be of two types—the action of shunts which occur both in relation to the main nodular vessel (pre-nodular shunts) and in the intranodular vessels (intranodular shunts); the intranodular shunts are determined by the occurrence of giant or sinusoidal vessels and are motivated by nervous and/or hormonal factors; (iii) vascular dilatation (in an area of epithelial activity), resulting in the redistribution of blood within a nodule and causing relative ischæmia of part of the nodule. The result of these vascular changes depends on both their duration and their intensity and may range from gradual atrophy of part of a nodule to frank necrosis of the whole structure. Secondary changes may be hæmorrhage within the nodule (comparable with changes in red infarcts seen elsewhere) and inflammatory changes (again comparable with those seen at the periphery of other infarcts). The author states that it is important to appreciate that these inflammatory changes may, with the passage of time, mask completely those changes resulting from the original vascular disturbances. If these changes are compared with those seen in infarcts elsewhere (for example, infarction or cortical necrosis of the kidney), they are seen to fall into line with other well known phenomena in the body and do not present as a special problem. While the majority of hæmorrhages into the thyroid gland are associated with the occurrence of necrosis, it is probable that the uncommon massive hæmorrhage may be due to factors other than those described in the preceding section. Obviously, a given nodule may present areas showing various stages in the above sequence of events, and in late cases it may be impossible to retrace the steps leading to the final state.

### Focal Myocytolysis of the Heart.

M. J. SCHLESINGER AND L. REINER (*Am. J. Path.*, May-June, 1955) state that focal myocytolysis of the heart is a miliary lesion which is characterized by a loss of muscular syncytium, preservation of the stroma, absence of inflammatory reaction and eventual fibrosis. Focal myocytolysis of the heart differs from a miliary infarct, which presents coagulative necrosis of muscle, often involves the stroma and has an active inflammatory reaction, but is not different in its eventual fibrosis. Focal myocytolysis of the heart is slower in evolution than in infarction. Focal myocytolysis of the heart is due to a lesser degree of metabolic imbalance than is miliary infarct. Focal myocytolysis of the heart is more common than miliary infarct.

### Molluscum Sebaceum.

C. D. CALNAN AND H. HABER (*J. Path. & Bact.*, January-April, 1955) state that the lesion of *molluscum sebaceum* starts with hyperplasia of a group of follicles, and this leads to hyperkeratosis and acanthosis with irregular downgrowth to the sweat-gland level. The hyperkeratotic masses of the follicles merge into one central mass to form a crater, whereas the acanthotic areas merge to constitute an irregular invasion of the cutis. In some cases the hyperplasia reaches such proportions that it is impossible to distinguish the individual follicles, and there appears a compact irregular mass consisting of merged follicles and apparently also of down-growing rete pegs. The sweat and sebaceous glands probably also take part in the formation of the lesion. In other words, the whole ectodermal part of the skin takes part in it. The corium itself provides the stroma, and hypertrophies by digitation of its papillæ; this leads to a kerato-acanthotic "papilloma". However, the lesion does not grow indefinitely; and as soon as it has, for some reason or another, spent its energy, it comes to a halt and is destroyed by the ensuing inflammatory reaction. This is clearly shown by the round-cell infiltration invading the tumour and by the many microabscesses. Since, in this process, the papillary body is destroyed, the lesion heals with scarring.

### Fat Emboli in Glomerular Capillaries of Choline-Deficient Rats and of Patients with Diabetic Glomerulosclerosis.

W. S. HARTROFT (*Am. J. Path.*, May-June, 1955) states that multiple fat emboli have been encountered in the glomerular capillaries of choline-deficient rats. Lesions induced by the emboli eventually simulate both focal and diffuse types of the Kimmelstiel-Wilson lesions in diabetic patients. Fat in the lumina of glomerular capillaries was found in frozen sections of kidneys of 75% of diabetic patients with Kimmelstiel-Wilson disease and of 15% of those without glomerular sclerosis. Vascular lipid not associated with glomerular disease was found in a small percentage of cases of alcoholic cirrhosis, but was absent in a variety of miscellaneous forms of renal disease unassociated with diabetes. Glomerular sclerosis in choline-deficient rats develops from embolic plugging of glomerular capillaries by fat. The authors describe the stages in the course of the development of the lesions and indicate their obstructive nature. They state that the liver is probably the source of the fat emboli in the rats. The source of fat plugs in glomerular capillary lumina of human diabetic patients is not obvious, but may be associated with the existence of hyperlipæmic states in the presence of elevated intraglomerular pressure. Evidence obtained from the present investigation and from those previously reported in the literature strongly suggests that glomerulosclerosis may result from capillary obstruction. It is possible that plugging of glomerular capillaries by fat, either entrapped as emboli or precipitated *in situ*, initiates the series of events that lead to formation of Kimmelstiel-Wilson lesions in diabetic patients.

### Hyaline Arteriolosclerosis in the Kidney.

J. P. SMITH (*J. Path. & Bact.*, January-April, 1955) has made a post-mortem study of the renal arterioles in 528 consecutive autopsy subjects comprising 266 normotensives, 226 hypertensives, 20 diabetics and 16 subjects of transient glycosuria. The anatomical features of the efferent arteriole are described, and the distribution of hyaline arteriosclerosis in both afferent and efferent arterioles is discussed. The author states that afferent and efferent arteriolosclerosis is found in normotensives, the incidence and severity being greater in males than in females and increasing with age in both sexes. Subjects in the early stages of hypertension show no more afferent arteriolosclerosis than do normotensives of similar ages; those in the late stages of hypertension show an increased incidence and severity of afferent arteriolosclerosis. Efferent arteriolosclerosis is not increased in subjects of hypertension but is increased in those of *diabetes mellitus*. In non-diabetics, there is a direct relationship between the incidence of efferent and

afferent sclerosis but no relationship between the severity of the two changes. The author concludes that hyaline arterio losclerosis of the renal arterioles is primarily an age change, and that sclerosis of the afferent arteriole is exacerbated by but not causative of benign essential hypertension. Histological analysis indicates that hyaline arteriolosclerosis is a degenerative change in the basement membrane and ground substance of the arteriole.

## MORPHOLOGY.

### The Tongue in Mastication and Deglutition.

SHAFIK ABD-EL-MALEK (*J. Anat.*, April, 1955) describes the movements of the tongue in mastication and deglutition. He states that in the preparatory stage the tongue is laid on the floor of the mouth and becomes trough-like. Next is the throwing stage, in which the anterior half, having collected the food-stuff, twists to one side and throws it on to the surface of the lower molars. Thereafter, in the guarding stage, the tongue remains twisted and, with the aid of the buccinator muscle, keeps the food between the grinding teeth. A sorting stage, in which insufficiently ground portions are collected and returned to the molars, then follows. When chewing is complete, the tongue makes side-to-side movements, mixing the food with saliva and rolling it into a bolus. In the first stage of deglutition the tip of the tongue is raised and pressed against the back of the front teeth, closing off the mouth and pharynx. For fluids the tongue here takes on a deep gutter-like form. Then the hyoid bone is pulled upwards and anteriorly, while the posterior part of the tongue is depressed; finally, the bolus is forced down and back by successive pressure by the tongue against the hard palate, from before backwards. In hemiplegia (say, of the right side), although the mandibular muscles of the left side are active, the muscles of the right side of the tongue are impaired, and the twisting and throwing action to place and keep the food between the molars is not possible; thus the food passes into the paralysed right cheek cavity, and what chewing the patient does is with the right molars, activated by the left-sided muscles.

### The Arterial Supply of the Human Prostate and Seminal Vesicles.

E. J. CLEGG (*J. Anat.*, April, 1955) describes in detail the blood supply to the human prostate and seminal vesicles, and compares the nomenclature used by previous authors. He finds that despite the contiguity of these organs, their blood supply has little in common. The prostate is supplied by the prostatic branch of the prostato-vesical artery on each side; this branch reaches the gland on its antero-lateral surface and passes down its lateral border, giving off fine twigs to the surface. It terminates as a bunch of small vessels which supply the pelvic floor and adjacent parts of the rectum and anal canal. Frequently another branch of the prostato-vesical artery supplies the posterior aspect of the seminal vesicle; Clegg gives it the name posterior vesicular artery. Occasionally this artery supplies part of the prostate, as do the superior rectal and vesiculo-deferential arteries. The vesiculo-deferential artery, usually arising from the site of origin of the umbilical artery from the internal iliac artery, is the constant source of supply to the seminal vesicle. It passes medially behind the supero-lateral border of the bladder, in front of the ureter (to which it gives branches) and continues medially to the lateral end of the seminal vesicle; here it divides into the vesical, deferential and anterior vesicular arteries (this last a new name proposed by Clegg). Tables are given showing the frequency of variations from the normal.

### Houston's Valves in the Human Embryo and Fœtus.

P. H. S. SILVER (*J. Anat.*, April, 1955) describes studies of the development of Houston's valves in the human embryo and fœtus. The valves make their appearance towards the end of the third month. The submucosa becomes thickened and is invaded successively by the circular and longitudinal muscle coats and finally by the perirectal connective tissue. The anterior and posterior parts of the longitudinal muscle take no part in the formation of the valves; thus, although the rectum appears as a straight tube from outside, internally it is a "zig zag" with the valves projecting in at the sides like baffle plates. When the rectum is fully distended with meconium the valves are taken up within the wall.

### The Innervation of Human Dentine.

R. COCKER AND J. M. HATTON (*J. Anat.*, April, 1955) report on the use of slow decalcification and a modified silver nitrate staining technique in sections of freshly extracted non-carious teeth. They report clear results which show the course of nerve axons from the pulp, between the odontoblasts, through the predentine, and for a considerable distance into the calcified dentine.

### The Mammalian Talus.

C. H. BARNETT (*J. Anat.*, April, 1955) discusses the anatomical and possible taxonomic significance of the degree of angulation of the talar neck of 98 mammalian limbs distributed over 48 species, as well as 35 human feet. The simplest type of talus has the long axis of its neck and both trochlear margins parallel to the long axis of the foot, which is itself at right angles to the axis of rotation at the ankle joint. This absence of angulation of the talar neck is characteristic of species with long narrow feet adapted for a cursorial gait. Medial deviation of the neck is found when the force is deviated within the foot towards its medial side, as in arboreal animals with a prehensile hallux, and also in wide feet in which the head of the talus lies to the medial side of the calcaneum, as in animals with a burrowing habit or a plantigrade gait. In species with a screw action at the trochlear surface the degree of angulation of the neck varies with differences in the pitch of the screw; these are the heavier animals, usually capable of jumping. In man, some arboreal primates and certain fossil species, the whole trochlea, and thus the axis of rotation of the ankle joint, is deviated with respect to the long axis of the foot. The author suggests that assessment of fossil tali on these lines might throw some light on the habits of extinct fauna.

### The Influence of Cortisone on the Development of Microglia.

E. J. FIELD (*J. Anat.*, April, 1955) reports on the stage at which microglia appears in the embryos of various animals, and on the influence of cortisone on the development of microglia in new-born rats. His conclusions are that mature microglia is present at early stages of embryonic development in the mouse, the rat, the sheep and man, and that foamy microglial cells become prominent late in development. He was not able to demonstrate sudanophilic material in these, and he found no conclusive evidence that they played any part in the process of myelination. Cortical islands of microglial cells are present in man and the sheep. Microglia probably arises from an early blood element, and is of mesodermal origin. Cortisone suppresses the formation of the neonatal microglial "fountains" of Hortega and causes considerable delay in myelination.

### Alkaline Phosphatase in Developing Teeth of Rats.

N. B. B. SYMONS (*J. Anat.*, April, 1955) reports on alkaline phosphatase activity, determined by the Gomori method, in the teeth of rat fœtuses aged from eighteen days to full term. He states that before odontoblasts or ameloblasts appear, activity is found in the stellate reticulum and *stratum intermedium*, and to a less extent in the dental papilla. After dentine and enamel have formed, activity is most intense in the *stratum intermedium*, less so in the external enamel epithelium of the incisors and in the pulp immediately deep to the odontoblasts, and still less in the stellate reticulum of the molars and in the odontoblasts. The ameloblasts show no activity until the enamel has matured, when it becomes intense. It is suggested that the alkaline phosphatase activity found in the very early stages of tooth development may be concerned in cell differentiation.

### Implantation of Epiphyseal Cartilage.

P. A. RING (*J. Anat.*, April, 1955) describes experiments to determine the possible polarity of epiphyseal cartilage. In young rabbits the epiphyseal cartilage of the distal end of the ulna, together with thin slices of the adjacent bone, was excised; in some it was replaced reversed, in some unreserved. The areas were followed by X-ray examinations and biopsies, and finally the animals were sacrificed. When the cartilage was replaced in its normal position, subsequent growth of bone was undisturbed, but after reversal the bony epiphysis became elongated — the cartilage retained its original polarity; bone growth was slow, and ceased after six to eight weeks.

# Special Article.

## THE CARE OF BLOOD DURING TRANSPORT AND IN HOSPITALS.[1]

THIS report consists of two parts. The first discusses the principles involved in the care and storage of blood. In the second part these principles are applied to hospital practice, and in addition certain recommendations are made to guide medical officers administering blood transfusions and to reduce the risks of accidents. It cannot be emphasized too strongly that blood transfusions can cause death. The same care should be taken in giving a blood transfusion as is taken in performing any surgical procedure. The risks of blood transfusion should always be borne in mind whenever the procedure is contemplated.

The principles defined in this memorandum are accepted in other countries, including the United Kingdom, the United States of America and Canada.

### PART I: PRINCIPLES OF BLOOD STORAGE.

During storage of blood it is essential that every effort be made to prevent (a) deterioration of the cellular and protein elements, (b) changes which may cause difficulty in administering the blood, and (c) infection with microorganisms.

#### Deterioration of Cellular and Protein Elements.

To the recipient the red cells are the most important cellular elements in the blood. The white cells and platelets are short-lived, except when special apparatus is used for collection of blood, and therefore need not be considered for practical purposes. Red cells are best preserved at a temperature between 1° and 4° C.[2] Freezing is dangerous.

Constancy of the temperature of storage above 0° C. is important. Evidence has been obtained that if the temperature of blood varies intermittently, the survival time of the red cells in the recipient is greatly diminished.

The rigid requirements for the storage of red cells must take precedence over factors designed to preserve the numerous protein components. In general the proteins are best preserved in the frozen state, but this is incompatible with survival of the red cells. However, with the exception of the antihæmophilic globulin, the proteins are fairly stable at temperatures above freezing point.

#### Changes which May Cause Difficulty in Administering Blood.

During storage the leucocytes and platelets settle on the top of the red cells and may be associated with a fibrinous deposit, which blocks filters of giving sets. This fibrinous deposit occurs only when the temperature of storage is between 0° and 6° C.,[2] but the change is irreversible. Storage above 6° C. does not produce the deposit.

#### Infection.

A small percentage of blood bottles will be contaminated even with the best techniques available. Unfortunately, some of the contaminating organisms grow at low temperatures (cryophilic and pseudocryophilic organisms), but the majority of contaminants multiply only at room or higher temperatures (mesothermic organisms). Active measures must therefore be taken (i) to prevent growth of unavoidable contaminants, and (ii) to prevent further contamination.

##### *Prevention of Growth.*

Because the majority of apparently unavoidable contaminants do not grow at low temperatures, it is imperative that blood be stored at a low temperature. In nearly every instance where patients have been given contaminated blood it has been found that the blood has been removed from the refrigerator and been allowed to remain at room or higher temperature for some hours prior to administration. This period at the higher temperature has permitted rapid multiplication of bacteria which were previously dormant. It follows therefore that blood should be administered to the patient as soon as possible. The transfusion should be commenced within one hour of removal of the blood from the refrigerator.

##### *Prevention of Further Contamination.*

During storage it is rarely if ever necessary to insert needles into bottles containing blood or to remove caps from the bottles. If, however, sampling is considered necessary, the blood from which the sample has been taken must be administered within one hour. If the blood is not given within the hour, the bottle should be labelled as unsafe for patients. There is evidence that the chances of contaminating stored blood are greatly increased even when the technique of sampling has been considered rigidly aseptic.

### PART II: CARE OF BLOOD DURING TRANSPORT AND IN HOSPITALS.

#### Care of Blood during Transport.

It is not always possible to maintain the desired constancy of temperature during transport. However, insulated ice boxes, with or without ice or chilled water inserts, reduce the rise of temperature during transport. Except in exceptional circumstances, such insulated boxes should be used whether the blood is delivered by road, air or rail, and blood should always be transported by the quickest method.

#### Storage of Blood in Hospitals.

##### *General.*

*Public Hospitals.*—Since blood has become widely and readily available there has been a tendency to overlook the possible hazards of transfusion therapy. What was once a rare procedure in a hospital is now an everyday occurrence. Organization has not kept pace with the increased usage and often has become somewhat haphazard. The responsibility for all phases of transfusion work in a hospital should rest with one of the more permanent members of the hospital staff who should be appointed as the transfusion officer. Unless this officer is available to implement and then supervise the various recommendations which follow, there can be little hope that the procedures will become part of hospital routine. The choice of transfusion officer must always be the concern of the hospital, but a few suggestions are offered: (a) In the large hospitals with a full-time pathologist, that officer is the obvious choice—or one of the pathologists if there is more than one on the staff of the hospital. (b) In the smaller hospitals with a visiting pathologist, the superintendent could be asked to undertake this duty with the advice of the visiting pathologist. (c) In the still smaller hospitals with no full-time superintendent, the matron might add the care of blood to her other duties with such local advice as is available.

*Private Hospitals.*—Except when they are associated with public hospitals it is difficult for private hospitals to obtain an efficient organization for blood transfusions. Detailed arrangements must be the responsibility of the medical practitioner administering the blood. However, experience has shown that the matron or deputy must also assist by drawing the attention of the medical practitioner to deficiencies in procedures such as the storage of blood or the return of unused blood.

##### *Refrigerators.*

Every hospital should have a refrigerator specially reserved for the storage of blood, serum and albumin. It should not be used for food or pathological specimens. The size will depend on the requirements of the hospital. Ideally the refrigerator should be fitted with an automatic temperature recording mechanism with the thermocouple placed at the point where the temperature should be lowest. Alternatively maximum and minimum thermometers should be used and both maximum and minimum temperatures recorded twice daily in a special book. The refrigerator should be adjusted to provide a temperature between 4° and 8° C. (39° and 46° F.)[1] and preferably should be fitted with an automatic defrosting mechanism. If this mechanism is not available, the refrigerator should be defrosted at frequent intervals in order to maintain constancy of temperature. Outdated blood and blood unfit for use should be stored separately from blood for transfusion.

##### *Central Storage.*

When blood is received at a hospital the bottle should be removed immediately from the carton or other form of packing used during transport and placed in the special

[1] Recommendations of the National Blood Transfusion Committee of the Australian Red Cross Society. They have been sent for publication by Sir Samuel Burston, Medical Adviser to the Society, and are commended to the attention of all medical practitioners.

[2] See Part II for recommended storage temperature in hospitals.

[1] This temperature represents a compromise to preserve the red cells without producing fibrinous deposits in white cell and platelet layer.

refrigerator. The transfusion officer should be responsible for seeing that this is organized efficiently. It is highly desirable that blood be not removed from the refrigerator until immediately before it is required for transfusion. If large hospitals find it essential to hold blood in operating theatres or labour wards, subsidiary refrigerators should be obtained specially for the purpose and must be subject to the same control as specified above.

*Handling of Blood in Hospitals.*

1. When dispersal of blood to subsidiary refrigerators is essential, the transfusion officer should be responsible either personally or by delegation to ensure that blood not used is returned to the central refrigerator each morning. Private hospitals should consult the Blood Transfusion Service each morning as to the disposal of unused blood. Transfusion accidents can follow administration of blood which has been allowed to remain in ward domestic refrigerators for long periods of time.

2. Blood should not be warmed prior to transfusion. It is safer to transfuse cold blood. In exceptional circumstances (for example, for exchange transfusions in infants) this may not apply. In such cases the bottle should be placed in warm water at a temperature not greater than 37° C. (98° F.). A thermometer must always be used to measure the temperature of the water.

3. Blood which has been frozen must never be used.

4. Concentrated red cells must not be stored and should always be used within twelve hours of preparation. The refrigerator used for holding concentrated red cells must have a constant temperature between 4° and 8° C.

BLOOD TRANSFUSION SHEET.

NAME OF PATIENT ..... Age ..... Sex ..... M.S.W. .....

WARD ..... HON. MED. OFFICER .....

R.M.O. ..... Date .....

Indication for transfusion .....

Amount of concentrated red cells required ..... bottles

Amount of whole blood required ..... bottles

Transfusion to be given on / / a.m.
/ / p.m.
Immediately

Has the patient ever received a transfusion of blood or plasma substitute?

If so, give details: Date ..... Material .....

Quantity ..... Group of blood .....

*To be completed by person performing tests*

Patient's blood group ..... Patient's Rh group .....

Patient's serum is compatible with:

| Bottle No. | Group of Pilot Bottle. | Bottle No. | Group of Pilot Bottle. |
|---|---|---|---|
| | | | |
| | | | |
| | | | |

Test for grouping and compatibility done by .....
(Signed)

THIS SHEET MUST BE CHECKED AGAINST THE LABEL ON THE BOTTLE OF BLOOD BEFORE BLOOD IS ADMINISTERED.

*To be completed by person administering blood*

Transfusion commenced at ..... a.m. on .....
..... p.m.

| Bottle No. | Commenced at | Note any adverse reaction |
|---|---|---|
| | | |
| | | |
| | | |
| | | |

Administered by .....
(Signed)

*Records.*

Two potential dangers in blood transfusion work are that (a) confusion may occur between the names of patients requiring transfusions, and (b) the results of cross-matching tests may not be accurately transmitted from the pathologist to the medical officer administering the transfusions. For these reasons and also because it is frequently necessary to trace the fate of a particular bottle of blood, accurate records are essential. It is suggested that the medical officer requiring blood should complete a Blood Tranfusion Sheet and send this sheet to the pathologist or responsible person who is to perform the cross-matching test.

The pathologist or responsible person should record the result of the cross-matching test on the Blood Transfusion Sheet and sign the sheet. The sheet should be returned to the patient's record. In addition, the pathologist should complete a compatibility label for every bottle of blood and attach it to the bottle. These labels should be removed from the bottle by the transfusion officer if the blood is not used within twenty-four hours.

COMPATIBILITY LABEL.

Bottle No. ..... Group .....

Compatible with .....
(Patient's name in full)

Patient's group .....

Date ..... Time .....

Does this label check with the patient's Blood Transfusion Sheet? .....

.....
(Signed)

A suggested Blood Transfusion Sheet (which may also serve as a record of the transfusion) and a suggested Compatibility Label are appended to this report. The exact type and size of the sheet will vary in different hospitals to conform with the hospital record system.

The Blood Transfusion Sheet and Compatibility Label should be completed on all occasions, even if the person who performs the cross-matching test also administers the blood.

It should be ensured that all members of the staffs of hospitals concerned in handling blood become fully cognizant of these recommendations.

# Clinico-Pathological Conferences.

## A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on October 18, 1955, DR. W. L. CALOV, honorary consulting physician, in the chair. The principal speaker was DR. G. E. BAUER, temporary honorary assistant physician of the hospital.

DR. W. L. CALOV: As Dr. Rose is unable to be present today, I have been asked to act as chairman. The speaker is Dr. Bauer, who, I believe, saw this patient during life, but is unaware of the end result. I will ask Dr. Bauer to open the discussion.

### Clinical History.

The following clinical history was presented.

The patient, a barman, aged sixty years, had been well until twelve months before admission to hospital, when he began to have attacks of giddiness, which increased in frequency to one nearly every day during the week prior to admission. During this time he also noticed increasing breathlessness on effort and suffered several attacks of breathlessness at night. In addition he experienced retrosternal pain and tightness brought on by exercise and relieved by resting for twenty minutes.

There were no complaints of headache, blackouts or ankle swelling, but he had had bleeding hæmorrhoids for twenty years and during the past year had suffered occasional attacks of "pain on micturition", for which he had been given tablets. He had also been taking digitalis before admission.

In the past he had had "rheumatism" at the age of fourteen years, but had otherwise been healthy. His family

history was not known. He smoked two ounces of tobacco each week and drank two pints of beer daily.

On examination the patient was found to be a rather thin, pale man in no respiratory distress while sitting up. His pulse was rather small and poorly sustained; it had a regular rhythm and a rate of 60 per minute. The blood pressure was 170 millimetres of mercury, systolic, and 100 millimetres, diastolic. The venous pressure was not raised. No peripheral œdema was present. The apex beat was not palpable, but the heart sounds were heard maximally in the axilla. There was a marked systolic thrill palpable over the right internal carotid artery, but no thrills were detected in the precordium. The first heart sound was loud in the axilla and the second sound soft in all areas. A harsh, blowing, grade 3 systolic murmur was present at the aortic area and was audible also at other areas, especially the mitral area.

The liver was firm, smooth and palpable one inch below the right costal margin. There were no abnormal signs in the chest. The respiratory rate was 20 per minute. A right inguinal herna was present. The urine had a specific gravity of 1·020; no albumin or sugar was present.

Chest X-ray examination showed some atherosclerosis of the aorta; the lungs appeared normal. A blood count showed a hæmoglobin value of 11·2 grammes per 100 millilitres of blood and a leucocyte count of 7400 per cubic millimetre, in normal proportions; platelets were plentiful. The results of Wassermann and Kahn tests were negative. The electrocardiogram showed changes suggesting a digitalis effect. The blood urea nitrogen content was 18 milligrammes *per centum* and the blood creatine content 1·0 milligrammes *per centum*.

Treatment consisted of a low salt diet and administration of digoxin 0·25 milligramme three times a day.

The patient complained of breathlessness and a stuffy feeling in the chest at night, although the respiratory rate was always 20 to 24 per minute. An isolated recording of blood pressure during his stay in hospital was 110 millimetres of mercury, systolic, and 79 millimetres, diastolic.

The symptoms gradually lessened, and the patient was discharged from hospital after two weeks. His hæmorrhoids were injected on three occasions in the out-patient department. He was readmitted to hospital six weeks later in a moribund condition with acute pulmonary œdema and died on the same day.

## Clinical Discussion.

DR. G. E. BAUER: I could not at first imagine why I should have been asked to perform at this conference. But I feel that I am quite experienced, having spent the last fortnight trying to find clues to win large sums of money, and tonight's problem is somewhat similar—if not to "find a word", at least to find a diagnosis.

The patient to be discussed tonight is a male of sixty years of age, who is a barman by profession. We straightaway think of certain hazards associated with that particular trade. It is quite true that the incidence of cirrhosis of the liver is some six to seven times as great in persons who habitually imbibe alcohol. We are told that he had been well till about twelve months before admission to hospital, when he began to have attacks of giddiness, which increased in frequency until about a week before admission, when he felt giddy just about every day. The symptom of giddiness embraces quite a number of systems: it might have something to do with the cardio-vascular system; equally, it might refer to the central nervous system, hæmopoietic system, ear nose and throat disease and many other things—we might leave that for the time being. During this time he also noticed increasing breathlessness on effort and suffered several attacks of breathlessness at night. I think we can definitely say that if a person gets breathless, dyspnœic, during exertion, and particularly if he has to sit up at night, he is suffering from attacks of left ventricular failure. We might as well consider the important conditions which can give rise to acute left ventricular failure.

The first thing which comes to mind is hypertensive cardio-vascular disease. Second on the list should be aortic valve disease, either incompetence or stenosis. The third condition we have to think of is mitral valve disease, either incompetence or stenosis. And the fourth condition is some form of ischæmic heart disease. In addition he experienced retrosternal pain and tightness brought on by exercise and relieved by resting for twenty minutes. Well, there are three important characteristics of cardiac pain. One is the site—retrosternal, though including a rather wide anatomical distribution, is a description which fits well for cardiac pain. Secondly, the character of the pain—and we are kindly enough told that he also experienced tightness, which is quite a classical description of cardiac pain. And thirdly, we are told that the pain comes on with exercise and disappears with rest, circumstances which are quite compatible with ischæmic heart pain. So I think we can say that this patient's symptoms comply with all three criteria for cardiac pain—site, character and circumstances which precipitate it and relieve it. When we think in terms of cardiac pain it is important that we should not just run to a diagnosis of coronary disease. Among the first things to be excluded when the history is suggestive of cardiac ischæmia are anæmia and thyrotoxicosis. Then we should think of aortic stenosis and then mitral stenosis. Recently there has appeared a very good article by Dr. Douglas Stuckey about the incidence of cardiac pain in mitral stenosis. Then we have the thought of aortic incompetence. If the history is one of prolonged chest pain indicative of cardiac infarction, we shall have to look for further confirmatory evidence in the history, physical examination and perhaps the electrocardiogram.

We have a few negative statements: he never complained of headache, blackouts or ankle swelling. He had had bleeding hæmorrhoids for twenty years. We might just bear this in mind, in view of the fact that he was a barman; bleeding hæmorrhoids may be the presenting symptom of cirrhosis of the liver. During the past year he suffered occasional attack of pain on micturition, for which he had been given tablets. We are not told whether he had had dysuria or nocturia. It may well have been a local condition of the lower part of the urinary tract. We will have to leave this for the time being. He had been given digitalis before admission.

The past history is negative except for a statement that he had had rheumatism at the age of fourteen years. We would like to know a little more about that—whether he remembers the features of this illness, whether he was admitted to hospital; in other words was it acute rheumatic fever which might have damaged the heart, or was it some other form of limb pains? The family history is not known. He smoked, and he stated that he drank two pints of beer daily, which probably means he drank at least double that quantity in his younger days.

So we come to the examination of the patient. We are told he was rather thin and pale, in no respiratory distress while sitting up. He might or might not be orthopnœic. The pulse was small and poorly sustained. Well, a small pulse may be of significance. We are not actually told that the pulse had the characteristics of an anacrotic pulse, which is characteristic of severe aortic stenosis. It was poorly sustained, but we are not told that it was of collapsing quality. It could still be collapsing though its volume was small. He was in regular rhythm. It is an important fact that here was a man in left ventricular failure without auricular fibrillation. The rate was 60 per minute; he had been digitalized. The blood pressure reading was 170 over 100 diastolic. Apparently the venous pressure was not raised; there was no peripheral œdema and no evidence of right-sided heart failure. Now comes a statement which is a little difficult to understand: the case report states that the apex beat was not palpable. We are first told that the man was thin, and he suffered, I believe, from left ventricular failure; so it is rather incongruous that the apex beat should not have been palpable. There must be some other reason for it; for example, he might suffer from emphysema, but there is no information on that. The heart sounds were maximal in the axilla; this statement, too, we will come back to at the end of the examination.

We are told that there was a marked systolic thrill over the right internal carotid artery, but no thrills were detected in the precordium. A thrill in the right carotid (I am not sure how to tell the difference between a thrill over the internal and external carotid branches) is a finding which may mean that a thrill is transmitted from the aortic valve; and a thrill in the aortic valve, which is not present here, means either aortic stenosis or aneurysm of the aorta. The thrill over the carotid, however, has to be interpreted with caution and is not of the same diagnostic significance as an aortic thrill itself. It is an important piece of evidence, but by no means absolute. The first heart sound is loud and the second soft in all areas. The soft second sound is also a finding in aortic stenosis. The second sound is made up of two components, closure of the pulmonary valve and closure of the aortic valve, the latter contributing the greater part of the noise. A classical sign of marked aortic stenosis is that the second sound, if at all present, is soft. He also had a harsh, blowing grade 3 murmur in the aortic area, but also audible in the mitral area. I take it that we talk in terms of four grades, in which case this murmur was a fairly loud one. A systolic murmur of that magnitude may be due to, again, aortic stenosis, an aneurysm of the

aorta, or merely calcification in the region of the aortic valve or first part of the aorta, a condition which has been described as aortic sclerosis. The fact that it is heard at the mitral area does not necessarily mean a separate lesion, because aortic systolic murmurs are very often heard at the mitral area, and it is not unknown for the murmur of aortic stenosis to be loudest at the apex. It may be so loud in that situation that there is an apical thrill due to aortic valvular disease.

There is not very much in the other systems. The liver is smooth and palpable one inch below the costal margin. That may be part of the cardio-vascular malady, or it may have something to do with our initial thought of cirrhosis. There were no abnormal signs in the chest, which makes emphysema unlikely. The respiratory rate was 20 per minute. And at last we have one positive diagnosis, a right inguinal hernia, if nothing else. The urine was normal.

We come now to two special investigations which were of some importance. The first thing we should look at is the chest X-ray film. It is a rather disappointing film; it does not help us terribly much. The heart shadow itself does not appear to be enlarged, perhaps a little full along the left border, but nothing very remarkable. There is nothing to suggest any left auricular enlargement and certainly no aneurysm of the aorta. On close inspection there is calcification in the aorta, which is not very remarkable at the age of sixty. Lung fields are not at all unusual, and I am sure we would all pass them without much comment. In passing you will notice that the ribs look normal and have no evidence of rib notching such as would remind us of coarctation. In view of this X-ray film one finds it difficult to know why the heart sounds were heard in the axilla. One other negative finding which I should mention is that we cannot see any calcification in the region of the aortic valve. We should not be very disappointed in that, because it is better seen under the fluoroscope, or it can be seen in special views and in particular tomography might be of help.

The next thing I would like to show you is the electrocardiogram. Remember that this patient was taking digitalis. The three standard leads show that the *QRS* complexes are taller in leads II and III, which is usually seen in a vertical heart, which we can check from the unipolar limb leads. We see *ST* and *T* wave changes in all three leads, which may be due to digitalis, though we cannot be quite positive that they do not represent some degree of ventricular hypertrophy. There is just one other point, which I notice now, and that is the notching of the *P* waves in lead II, which is suggestive of auricular enlargement. In the unipolar limb leads we find left ventricular pattern in *aVF*—in other words this is a vertically placed heart. The six precordial leads show progressive increase in the *R* waves across the chest and *ST* depression which may be due to digitalis, but it may also be due to left ventricular hypertrophy in a vertical heart. Left ventricular hypertrophy in a vertically placed heart is not very unusual. Hypertension and aortic incompetence usually cause left ventricular hypertrophy with a horizontally placed heart, but aortic stenosis does just that peculiar thing, giving hypertrophy in a vertically placed heart.

Blood count shows a mild anæmia with a normal white cell count. The Wassermann and Kahn reactions are negative. It is nice to know that. We are not very worried about aortic incompetence, not having heard a diastolic murmur, but that murmur may, at times, be missed. Blood urea nitrogen and creatinine contents are within normal limits.

The rest of the story is very simple. He was treated with low salt diet and digoxin. There are three main principles in the treatment of heart failure: digitalis in some form or other, sodium restriction and mercurial diuretics. It was apparently thought not necessary at that time to give him mercurial diuretics. In hospital he was still breathless and felt stuffy in the chest at night time, which still sounds like pulmonary congestion, but his respiratory rate was always 20 to 24 per minute. Symptoms improved, and he was discharged after being in hospital for a fortnight. His hæmorrhoids were injected, and he was readmitted six weeks later, moribund, and he died on admission of pulmonary œdema. So that is the whole story, and we might just as well now see which of these conditions are still in the running.

I think hypertensive cardio-vascular disease need not be very seriously considered. The one reading I have gives a diastolic pressure of 100 millimetres. The heart does not look hypertensive, and I am prepared to dismiss hypertension without further discussion. Aortic incompetence I think we can dismiss on three grounds. First of all, we could not hear the murmur of aortic incompetence. Secondly, there were no peripheral signs of incompetence. Thirdly, there was a negative Wassermann reaction. Aortic stenosis I will come back to, because I think that is very much on the cards. Mitral valve disease—we have a history of rheumatism, but there is very little in the physical examination or in the X-ray film to suggest mitral valve disease. We have the mitral systolic murmur, but we can explain that on other grounds. We must, however, remember the notching of the *P* waves in the electrocardiogram.

The two main conditions to be discussed are ischæmic heart disease and aortic stenosis. As far as ischæmic heart disease is concerned, we can be quite certain on account of the history and the electrocardiogram that, at least on the first admission, he did not suffer from cardiac infarction.

I would like to say something about aortic stenosis. The history is quite compatible with such a diagnosis. It is characteristic for aortic stenosis to remain well compensated till late in life. The average age when symptoms appear is about fifty-five; but once symptoms have appeared the outlook is usually very grave, and in the majority of cases not more than twelve to eighteen months elapse before death occurs. Such was exactly the case in this patient. Men, of course, more frequently suffer from aortic stenosis than women. That goes for all diseases of the aortic valve and the aorta.

The symptoms of aortic stenosis usually fall into three groups. First of all, there may be symptoms of left ventricular failure. Secondly, symptoms may be due to cardiac pain or rest pain like that of coronary insufficiency. And thirdly, there is a peculiar symptom of syncope and giddiness which is characteristic of aortic stenosis. Usually the syncope is exertional, but it may occur even at rest. If very severe it may result in actual loss of consciousness, or the patient may complain simply of giddiness, as was the case with this patient.

The appearance of the patient has been described by Paul Wood as resembling Dresden china; so it is rather interesting that our patient is said to have been pale and thin, though I must say that when I first saw him he did not remind me of Dresden china. The pulse of aortic stenosis is usually small if the stenosis is severe and characteristically gives an anacrotic tracing with a slow upstroke, a plateau and a slow downstroke. Usually less than 10% of patients develop auricular fibrillation, which is very different from mitral valve disease. The blood pressure in severe stenosis is said to be low, but that is by no means absolute, and the reading of 170/100 does not necessarily put us off the diagnosis. The apex beat is usually of left ventricular type, but in a vertically placed heart there would not be much outward displacement of the apex, unlike the findings in hypertensive disease or aortic incompetence. This, too, is rather reminiscent of our patient. In many cases, but not all, you find a thrill in the aortic area. Up till a few years ago a thrill was regarded as essential to the diagnosis of aortic stenosis, but we now know that more than half the cases will be missed if this sign is insisted upon. In any case we have a thrill in the carotid, which may be significant. With regard to the heart sounds, the second sound is diminished, owing to the inability of the aortic valve to vibrate and to contribute to the second sound. All cases have a loud systolic murmur, most commonly maximal in the aortic area, but it may be widely conducted, and in some instances it is loudest at the apex. The murmur is usually harsh, as it was in our patient. The loudness of the murmur does not bear a direct relationship to the severity of the stenosis. We know that when the stenosis is very tight—say, less than half a square centimetre aperature—the murmur may diminish in intensity. And the other thing to remember is that when the heart fails, the murmur may decrease in intensity and actually disappear. One patient I have seen with this condition was admitted to hospital in terminal heart failure without any murmur at all, though previously he had had a loud one with a thrill. So the loudest murmurs are those with a moderate stenosis and not those with a very tight or a very slight stenosis. The other thing about the murmur is its timing. It usually follows straight after the first heart sound, takes on a *crescendo* quality and then disappears towards the first component on the second heart sound. Phonocardiographically it has a very characteristic and diagnostic pattern: it is diamond-shaped. It is loudest in mid-systole, beginning straight after the first heart sound and continuing to the first component of the second heart sound. This is a very nice differential diagnostic point. In pulmonary stenosis the murmur may continue past the aortic component of the second sound and go on to the pulmonary component. In mitral incompetence the murmur is plateau in shape rather than diamond-shaped, and this investigation, in doubtful cases, may well be useful.

The electrocardiogram, as I mentioned, usually shows sinus rhythm and left ventricular hypertrophy in a vertically

placed heart. In an electrocardiogram department one can sometimes pick suspicious cases on these features alone. X-ray examination usually shows some degree of left ventricular hypertrophy. Fluoroscopy will often reveal some dilatation of the ascending aorta, very similar in pathogenesis to the post-stenotic dilatation found in congenital pulmonary stenosis. There may be calcification in the aortic valve, and the place to look for that is in the middle of the heart shadow. If in the antero-posterior view the heart shadow is divided into three segments, aortic valve calcification will fall in the middle third and mitral valve calcification will be in the left hand third. In the first oblique view, the right anterior oblique, a line should be taken from the junction of ventricle with auricle backwards and downwards at an angle of 45°; then mitral valve calcification will be below the line, and aortic valve calcification above the line. Lastly, I think tomography might be of help in showing this particular calcification.

In considering aortic stenosis I think we have to say a few words about the ætiology of the condition. There are three ætiological factors to be considered. It may be a congenital lesion. Congenital aortic stenosis was regarded as rare until several years ago, but now more and more reports are appearing, and it is becoming obvious that many cases of asymptomatic aortic stenosis which are picked up in later life caused a murmur in early life, in infancy, and are probably congenital in origin. The second ætiological factor is rheumatic heart disease, and in 1947 Karsner and Koletsky wrote a monograph where they tried to prove that practically every case of aortic stenosis was due to rheumatic fever. In our case we had a history of rheumatism, not a good history, but it has to be considered. Pathologically speaking, we ought to be able to distinguish this from the third group, the arteriosclerotic stenosis. In rheumatic stenosis there is adhesion of the cusps and calcification of the free end of the cusps with stenosis due to cusp adhesion and narrowing of the aortic orifice. In arteriosclerotic aortic stenosis the stenosis is due more to valve immobility from the deposition of calcium in the sinus of Valsalva. The differentiation is not only of great pathological interest, but is also becoming of great surgical importance now that aortic stenosis has become one of the surgical cardiac maladies. The surgeon is very interested in the state of the valve before actually operating upon it.

Finally, what is our conclusion on this particular patient? I think the diagnosis must rest with aortic stenosis, though I cannot quite convince myself that it might not be ischæmic after all. So I would like to suggest that this man died with congestive cardiac failure and pulmonary œdema due to aortic stenosis, possibly rheumatic, in which case we will not be surprised to find some pathological change in the mitral valve, although it gave rise to no signs, with the exception of the electrocardiogram, of mitral valve disease. Or it was due to arteriosclerotic aortic disease. Way back in our mind we still think of some liver pathology, and one thing we are sure of is that he had a right inguinal hernia. Finally, the last two or three patients with aortic stenosis who came to autopsy surprised me by having gross pulmonary infarction, quite unsuspected in life, without any cough, hæmoptysis, fever or other suggestive symptoms: I will just keep that in the back of my mind.

DR. CALOV: I must thank Dr. Bauer for a very full and learned discussion of the case. It is now open for comment. Would Dr. Grattan-Smith like to tell us anything about the X-ray film? Would you consider there could be left ventricular hypertrophy even though it is a narrow heart?

DR. P. GRATTAN-SMITH: Well, I think it is possible, but I certainly would not diagnose it just on that film.

DR. G. MICHELL: I admired Dr. Bauer's masterly analysis of the case, and I agree it is probably aortic valvular disease most likely of rheumatic origin.

DR. P. FRANCIS: I would like to point out that there was one other reading of blood pressure which did not appear on the history sheet given to Dr. Bauer. It was 110/70, and tends to support Dr. Bauer's thesis.

DR. CALOV: Are there any pathologists who would like to comment?

DR. E. HIRST: I am puzzled by two of the findings. In my ignorance I would think that the blood pressure reading and the normal-looking X-ray film were inconsistent with the diagnosis of aortic stenosis. I would then conclude that the diagnosis is arteriosclerotic heart disease with a severe degree of atheroma of the coronary vessels.

DR. CALOV: I suppose I should say something. First of all I would suggest that the lung fields do show congestion in the X-ray film. I think that Dr. Hirst has put forward some very strong points against left ventricular failure. On the other hand, I do believe those markings are increased, and it may well have been that a film taken at another time, when the patient was in greater distress, might have shown more congestion and perhaps some dilatation. It would seem to me that as this is a long heart, he may well have had some left ventricular hypertrophy, although it could not have been very great. I would suggest that the heart may have been turned into the left hemithorax for some reason or other—I wondered whether he had a scoliosis, but I cannot see any sign of it in the film. Finally, as to the cause of death, it could have been an infarction, sudden left ventricular failure for reasons that I do not understand or (as Dr. Bauer suggested, and I am very glad he mentioned it) pulmonary infarction: I was thinking he may have had some complication from his hæmorrhoids or from the treatment. Do you wish to reply, Dr. Bauer?

DR. BAUER: I am rather relieved to hear of the other reading of blood pressure, which is more in keeping with what we expect of aortic stenosis. High blood pressure does not exclude the diagnosis. Left ventricular failure in this condition very often is episodic and, as Dr. Calov mentioned, X-ray films taken at different times may show great variations.

### Autopsy Report.

DR. A. A. PALMER presented the following abstract of the post-mortem report:

The body was that of a middle-aged male in fair nutrition. The weight was 125 pounds.

Pleural cavities: There were six ounces of clear fluid in each pleural cavity.

Lungs (right, 36 ounces; left, 29 ounces): Both lungs were very œdematous.

Heart (16 ounces) (normal 9½ to 13 ounces): The pericardium was normal. There was hypertrophy but no dilatation of the left ventricle; the right atrium was somewhat dilated. The right ventricle, tricuspid and pulmonary valves were normal. The mitral valve was calcified and stenosed, admitting one finger. The aortic valve showed gross calcification with rigid cusps causing stenosis and incompetence. The orifice was slit-shaped and admitted a small pencil. The aorta showed slight atheroma, and the coronary arteries showed atheroma with calcification but no narrowing.

Liver: The liver was of normal weight (62 ounces) and showed only slight congestion.

Other organs: The spleen, kidneys, pancreas and adrenals were normal.

Rectum: The rectum was thickened and rubbery, and the perirectal fat showed scarring with small cystic regions of necrosis.

*Miscroscopic Examination.*

Heart: There are elongated foci of infiltration with cardiac histiocytes, lymphocytes and occasionally eosinophils. In some of these lesions there are small regions of necrosis. No giant cells are present, but the lesions are suggestive of Aschoff bodies.

Rectum: There is striking infiltration with eosinophil leucocytes and lymphocytes throughout the rectal wall and in the perirectal fat the inflammation is sometimes accompanied by necrosis.

### Diagnosis.

Rheumatic heart disease with mitral and aortic stenosis; pulmonary œdema; inflammation of the rectum.

## British Medical Association News.

### SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held in the Department of Pathology, University of Melbourne, on September 28, 1955.

### The Basic Mechanisms of Psychosomatic Disorder.

DR. BRUCE ROBINSON presented a demonstration setting out basic mechanisms involved in psychosomatic disorder. Physiological, anatomical and psychological components

were discussed, and means were suggested by which these could be correlated to produce the resultant disturbance or disease.

### Large Sections Illustrating Lung Pathology.

DR. K. H. MCLEAN presented thin sections of whole lungs, prepared according to the method of Gough and Wentworth, as paper mounts and as wet mounts in "Perspex" jars. The sections illustrated various common pathological conditions of the lungs.

### Bronchial Changes in Emphysema.

Dr. McLean also discussed the pathogenesis of emphysema under the following headings: (*a*) the evidence for a localized basic lesion producing all types of emphysema; (*b*) the unsatisfactory nature of those theories of ætiology of emphysema that have been presented; (*c*) the constant association of ectasia of the bronchi and bronchioles with emphysema; (*d*) the extensive collateral ventilation in developed emphysema; (*e*) a summary of the technical problems involved in this investigation—basically that of three-dimensional reconstruction of 500 to 1000 8$\mu$ serial sections.

### Idiopathic Pulmonary Hæmosiderosis.

The pathology of a recent case of idiopathic pulmonary hæmosiderosis formed the basis of a demonstration by DR. L. I. TAFT. He said that the natural history of the disease was one of recurrent "idiopathic" acute intrapulmonary hæmorrhages occurring in childhood. He showed that with each attack siderophages appeared in the alveoli, with interstitial inflammation, deposition of hæmosiderin in interstitial tissue, subpleural lymphatics and hilar lymph nodes, and progressive diffuse pulmonary fibrosis. Death occurred from respiratory embarrassment, anæmia and heart failure in an acute attack. The radiological, macroscopic and microscopic changes in the lungs were displayed.

### Morphogenesis of Renal Cysts in a Stillborn Infant.

DR. DORA BIALOSTOCK showed photographs of a specimen of renal cysts in a stillborn infant with explanatory legends covering macroscopic, microscopic and microdissection parts of its investigation. Stages in cyst formation were followed in the histological sections. Microdissected material was subdivided into the appearance of normal nephrons, the types of cyst, glomerular and tubular, and two types of abnormal nephrons associated with cystic glomerular elements. The photographs showed that cyst formation could occur anywhere along the length of the nephron. A short note on the technique was included emphasizing the difficulty in assessing correct maceration of the specimen prior to dissection.

### Atheroma of the Aorta.

DR. J. D. HICKS and DR. R. MCD. ANDERSON presented a review of the literature on atheroma of the aorta with a demonstration of specimens, ranging from lipoid deposits in the intima of the aorta to gross atherosclerotic lesions complicated by aneurysm or massive thrombosis.

### Tumours of Lymphoid Tissue.

DR. I. EPSTEIN demonstrated tumours of lymphoid tissue showing four main histological patterns—namely, lymphosarcoma, reticulosarcoma, Hodgkin's disease and follicular lymphoma. Photomicrographs of each pattern were shown. Representative macroscopic specimens of involvement of lymph nodes, spleen, lung, kidney and alimentary canal were on view. The age and sex incidence of the tumours and the clinical manifestations were referred to.

### Dissecting Aneurysm of the Aorta.

DR. J. H. W. BIRRELL and DR. J. V. HURLEY demonstrated the clinical and pathological features of 104 cases of dissecting aneurysm of the aorta collected from the records of the Melbourne teaching hospitals. It is proposed to publish the details of these cases in the near future.

### Skeletal Changes in Lathyrism.

DR. K. W. MILLS gave a demonstration of the skeletal changes produced by feeding the sweet pea (*Lathyrus odoratus*) to young rats. The essential deformities shown by X-ray film were kyphoscoliosis, epiphyseal changes about the large joints and new bone formation along the shafts of the long bones. Photographs of the histological appearances were presented, displaying deformity and altered maturation of the cartilage, columns in the epiphysis, degeneration of the intervertebral disks of the spine with changes leading to kyphoscoliosis and a young fibrous tissue reaction of periosteum, which later progressed to calcification.

### Aortic Changes in Lathyrism.

By a series of photographs DR. D. W. MENZIES demonstrated the effect of ingested sweet pea seed on the aorta of rats. He said that the primary lesion appeared to be a disruption of interlaminar reticulum by a ground substance disturbance as yet not fully identified. That was followed by intramural hæmorrhage, usually tracking between the adventitia and media, but occasionally producing true medial dissection. The relation of the experimental effects to human dissecting aneurysm was not clear.

### Medico-Legal Miscellany.

Specimens and details of cases of medico-legal interest were presented by DR. J. H. W. BIRRELL. They included the following: knife and gun-shot wounds and other results of trauma; cases of heart disease, including Marfan's syndrome; a rare case of asphyxia due to an idiopathic stenosed calcified larynx; a case of death on the operating table from acute pulmonary œdema associated with phæochromocytoma.

### Some Aspects of Fatty Livers.

Some aspects of the production of fatty livers in rats by choline and methionine deficient diets were presented by DR. M. G. WHITESIDE with consideration of the biochemistry and the dietary factors involved. He demonstrated examples of fatty cysts which developed in livers after prolonged periods on the diet, the resultant fat embolism in the lungs from those cysts, and features of the early development of portal cirrhosis. It was also shown that no fatty change or cirrhosis occurred in the livers of newborn rats when the mother was kept choline deficient.

### Chemical Carcinogens.

The chemical structure and sites of action of various groups of chemical carcinogens were demonstrated by MR. N. MCCALLUM and DR. P. HUGHES. They said that in recent years over 1000 compounds had been tested for carcinogenic activity; about 25% of them were found to be active. The compounds could be divided, according to their chemical structure, into groups such as the polycyclic aromatic hydrocarbons, amino-azo dyes, amino-compounds and biological alkylating agents.

### Causes of Hæmorrhage from Female Genital Tract.

DR. C. R. GREEN demonstrated a number of specimens illustrating some conditions associated with hæmorrhage from the female genital tract. They included: benign and malignant lesions of the vulva, vagina, cervix and body of the uterus; endometrial tuberculosis and inflammations associated with incomplete abortions and contraception; granulosa cell tumours of the ovary; hydatidiform mole and chorion carcinoma; and ectopic and intrauterine pregnancy in which hæmorrhage had occurred.

### Specimens from the Christian Medical College, Vellore, India.

DR. E. W. GAULT demonstrated examples of conditions seen in the pathology department of the Christian Medical College, Vellore, South India. They included the following: (i) Rhinosporidiosis, a fungus infection which mainly affected the nose but also involved the lachrymal apparatus, eye, skin, lung and genitals. (ii) Silver staining for nerve endings in the skin and for spirochætes in tissue section from an ulcer in the small intestine in a case of congenital syphilis. (iii) Changes in nerves in the skin from leprosy patients. (iv) Epithelioma of the oral cavity, a condition associated with the chewing of lime and tobacco.

The demonstrations were illustrated by clinical and macroscopic pictures and photomicrographs.

### Lung Cancer.

A demonstration was arranged to correlate the clinical story, X-ray films and pathological specimens in nine cases of lung cancer with a normal lung and X-ray film for comparison. Mechanical extraction of carcinogens from tobacco was on display, together with pictures showing the method of smear diagnosis for cancer cells.

# Out of the Past.

*In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.*

## NATIVE AILMENTS.[1]

[From "An Historical Journal of the Transactions at Port Jackson and Norfolk Island", by John Hunter, Esq., Port Captain in His Majesty's Navy, London, 1793.]

AUGUST, 1791: A disorder had frequently been seen amongst the natives which had the same appearance as the itch and yielded to the same remedies: it was now so common, that nearly the whole of them were infected with it, and several boys were cured at the hospital by rubbing in of brimstone. Benelong was a perfect Lazarus, and though he was easily persuaded to go to the hospital and rub himself, yet it was not possible to make him stay there till he was cured.

There people pay little attention to wounds, and even those which by the faculty are deemed dangerous, do not seem to require the common attention of closing the lips of the wound and keeping it clean: this shews they must be of a most excellent habit of body.

# Correspondence.

## A DOCTOR WANTED FOR FIJI.

SIR: I write to you with regard to the need for a medical practitioner in the Ba area on the island of Viti-Levu, the main island of the Fiji group.

For some years the Methodist Church has conducted a hospital for Indian women and children at Ba, and a few months ago the medical officer of this institution was obliged to resign. The hospital has 55 beds and has a staff of Indian trainee nurses led by three Sisters from Australia. The hospital is also a training centre for Indian nurses, and the medical officer assists in this work.

Ba is situated in one of the most thickly populated areas of Fiji, and the 40,000 people on that side of the island are at present only partially served medically. The practitioner appointed to the church hospital at Ba would be able to engage in private practice by arrangement with the hospital committee.

The need at Ba is urgent, and I would be very glad to discuss with any doctor who would be interested further details concerning the work at the hospital, remuneration, accommodation *et cetera*.

Yours, etc.,

C. F. GRIBBLE,
General Secretary, Methodist Overseas Missions.

139 Castlereagh Street,
Sydney,
January 12, 1955.

## THE BRITISH MEDICAL ASSOCIATION AND ITS MEMBERS.

SIR: Before we join Dr. Munro Alexander in his clarion call to the profession "to wake up to itself", let us remember other facets of medicine outside its practice. Worthy indeed is its Area, State and Federal organization, trying to reduce to a few voices the "babel" of thousands, traditionally free of thought and speech, in a community subjected to advancing bureaucracy.

But worthy also are the claims of Post-Graduate Committees, of the Colleges and Special Sections—the claims of university teaching and honorary service, the claims of research and, beyond that, the claims of service to the community on local councils, political and charitable organizations or in military duties, and last but not least the claims of a man's right to indulge some of his leisure in the pursuit of culture, of sport and of personal happiness.

The profession should not be attacked for its "apathy" in a particular sphere because that particular sphere feels it has a prior claim on its members. A good councillor rightly earns the gratitude of the profession for his arduous and time-consuming duties on our behalf; but likewise, so should those members who by research, by teaching, by cultural pursuits or community service contribute to our professional and social status.

In times of stress or threat to our professional way of life there was no evidence in the past of "apathy". In the lulls between storms the "apparent" neglect of Association matters persists just as long as members feel that local councils and committees justify their trust, and that they are represented by conscientious, energetic and worthy members such as Dr. Alexander.

Yours, etc.,

JOHN SANDS.

75 Dowling Street,
Kensington,
New South Wales.
January 10, 1956.

## THE PRESENT POSITION OF THE ARMY MEDICAL SERVICES IN AUSTRALIA: A NATIONAL SCANDAL.

SIR: The verbal outbreak of Dr. Benwell in your issue of December 17 prompts comment.

His excuses for the lack of medical officers in the army seem to have been accepted already by many. However, they are excuses and as such are as valuable as the paper on which they are written. His use of such words as "moral cowards", "defeatists", "idealists" are meaningless, and he has only lengthened the weakness of his argument by using them. He says "a core of military age individuals is not shirking its moral obligations, but rather is striving to find where its obligations truly lie". Their striving does not appear to have been very energetic, and their obligations appear to have been an automatic choice.

How quickly Dr. Benwell has forgotten the past, although he talks of simple deductions from contemporary history—surely he has read more than the daily paper? He indicates that as nobody can win another war why worry about it! The statesmen of the world have seen fit to attempt the prevention of a third world war by using their experiences of the last two. They feel that only a show of military strength—call it military preparedness if you like—can hope to avoid an open conflict. We put these men into power, and we must support their decision. They have at least avoided a major war for ten years.

Does Dr. Benwell think that doctors are such outstanding men both mentally and physically that they could easily fight another war if required without any training? He admits the services are pointedly ignored by the newly qualified men, be it on moral or financial grounds. Is our obligation to our country any less than that to our patients? The Army Medical Services do not want "doctors" as such, they want men, and as long as people like Dr. Benwell attempt to defend the "doctors" with irrelevant excuses the army will remain understaffed. Perhaps that is a good thing. Those who do come forward will have understood the nonsense he writes.

Yours, etc.,

C. STUART.

179B St. George's Terrace,
Perth,
December 28, 1955.

SIR: On November 5, 1955, you published an editorial dealing with the shortage of medical officers in the Army Medical Services in Australia. By December 17, 1955, the date on which you published, with noteworthy impartiality (or perhaps indifference), a letter from Dr. W. S. Benwell castigating you and the Director-General of Medical Services, all vacancies in the volunteer medical units in Eastern Command had been filled, and there was every prospect that the Permanent Army vacancies would shortly be filled also. It would seem that the men with the long list of negative virtues whom Dr. Benwell gratuitously defended with a mass of woolly philosophy, as soon as they became aware of the need, hastened to offer their services.

Unrealistic idealism, even though expounded with the aid of every known fallacy of logic, can do much harm. There does exist today an ominous threat of disastrous war. By a curious coincidence, at about the same time that Dr. Benwell was writing that refusal to join the Army Medical

[1] From the original in the Mitchell Library, Sydney.

Services would prevent war, the "biggest ever" H-bomb was being exploded in Russia. Only a high standard of military preparedness has prevented the threat of war from becoming an actuality. While armed forces exist, for whatever reason, there must be medical services. They at any rate are not belligerent—they bring succour to the victims of war; they are playing an ever greater part, and, as things are today, they are the only services receiving training in methods of coping with atomic explosions. Surely it is wiser to make whatever preparation is possible for a threatened emergency than to decry such preparedness as futile and provocative.

Yours, etc.,

96 Woodland Street,
Balgowlah,
New South Wales.
January 5, 1955.

CARL GUNTHER.

## ANOMALIES IN THE SCHEDULES OF COMMONWEALTH MEDICAL BENEFITS.

SIR: There are so many anomalies in the schedules of Commonwealth Medical Benefits that one expected to see changes as the schedules were revised. However, a new copy has just been received, dated January, 1956, in which all the old faults are maintained. I can comment only on the orthopædic items. In some cases the benefit does not bear any relation to the extent of the service performed; in others benefits are mentioned and apparently available for services which could only be regarded as unnecessary or even meddlesome. I will mention only a few examples.

The correction of *hallux valgus* in both feet, which is technically only slightly more difficult and longer than the amputation of two toes, is quoted at £9 7s. 6d. This places it well ahead of the treatment of a fractured femur, an osteotomy of the spine or an "arthrectomy of the shoulder", all £7 10s. In contrast, the suture of a nerve trunk, a skilful procedure which usually takes over an hour in expert hands and in which the future function of a limb is usually at stake, is worth only £2 12s. 6d. In comparison, it is less important but nevertheless interesting to note that an amputation of the little toe (£1 17s. 6d.) is rated higher than an amputation of the thumb or a finger (£1 10s.).

I had forgotten Sever's disease, but found in a text-book that it is, of course, an apophysitis of the *os calcis*. Why anyone should want to manipulate such a case, however, is difficult to understand; the same applies to Perthes's, Köhler's, Kienbock's and Scheuermann's diseases. There is every reason to believe that in most cases of this type a manipulation would actually do harm.

The importance of the Commonwealth Medical Benefit Schedules need not be stressed. They are likely to influence every practitioner in Australia. As they stand they are quite unfair, and they suggest that the standard of treatment in at least one branch of surgery in this country is both outmoded and irregular.

Yours, etc.,

137 Macquarie Street,
Sydney,
December 20, 1955.

HUGH C. BARRY.

## FUNNEL CHEST: REPORT OF A SERIES OF ONE HUNDRED CASES.

SIR: Dr. Russell Howard's review of his experiences in the treatment of funnel chest (M. J. AUSTRALIA, December 31, 1955) and his comments should focus the attention of the profession on this interesting condition.

Recently I discussed the management of this condition with Mr. E. F. Chin (Consulting Thoracic Surgeon, South West Region, England), whose work in this field has been rewarded by his appointment as a Hunterian Professor in the Royal College of Surgeons. Mr. Chin's contentions concerning management closely follow those of Dr. Howard, except in minor technical details.

I have operated on 17 cases of funnel chest between the ages of fourteen months and forty years. The only unsuccessful ones were the two youngest (fourteen months and twenty-six months), in which the procedure advocated by Brown (1939) and Lester (1950), and referred to by Dr. Howard in his article, was carried out. I think it is now generally agreed that this procedure is useless. In my experience the attachments to the posterior surface of the lower end of the sternum are variable, and in fact sometimes non-existent; since it is not the fundamental cause of the deformity, as was originally thought, it is no wonder that this simple procedure is ineffective.

Dr. Howard apparently uses a transverse incision in all cases. On his recommendation I have used the transverse incision and have found it to be ideal for children, but too cumbersome for adults, unless the deformity is localized to below the third cartilage.

In selecting cases for operation I consider that the retraction of the lower sternum on inspiration is a strong indication. I agree that operation should be withheld until the child is over four years of age, but the results in the older age groups have been so encouraging that the upper age limit has been generally extended to forty years.

According to Mr. Chin, operation should be avoided in the large shallow "saucer-like" deformity, because of the likelihood of recurrence. Conversely the best results are obtained where the deformity is deep and relatively localized.

It is claimed by some clinicians that the benefits of the operation are purely psychological, but I am satisfied from the observations of patients, parents, school teachers *et cetera* that the improvement is physical as well as cosmetic and psychological.

Yours, etc.,

80 Brougham Place,
North Adelaide,
January 4, 1956.

H. D'ARCY SUTHERLAND.

## ADVERTISEMENTS AND SAMPLES FROM DRUG HOUSES.

SIR: As an avid student of your most knowledgeable journal, may I, as a layman, be allowed a few lines, not to stretch out a controversy, but rather to close it on a note of defence and to give thanks in particular to your most recent correspondent, Dr. W. Alexander Dunn, for his kindly words on "travellers". Having travelled for a leading drug house in Queensland, New South Wales and now Western Australia, I feel in a position to write in defence of my fellow travellers—who, after all, have a job to do—and say that for every one physician who makes our approach difficult there are twenty who are sufficiently interested in finding out "what's new?".

Lest it be thought that travellers are more vitally interested in medicines than medicine, let me conclude with an illustration of how we have played our part here in Western Australia. Upon the formation of an appeal for funds to found the Medical School of Western Australia, one of the first committees to be formed voluntarily—and of which the writer is a member—was composed of travellers from the various drug houses. Backed by our fellows the committee have worked unceasingly to raise funds for this appeal and will be responsible in raising at least a four figure sum so that Western Australia can cradle future men of medicine.

I am sufficiently proud of those fellows who travel my road to say of them that should this or a similar opportunity present itself to help medicine in any other State they would welcome it.

Yours, etc.,

Box H570,
G.P.O.,
Perth.
January 7, 1956.

GORDON A. FOX.

# The Royal Australasian College of Physicians.

## VICTORIAN STATE COMMITTEE.

THE Victorian Fellows and Members of The Royal Australasian College of Physicians will hold a scientific meeting at the Ballarat and District Base Hospital on Saturday, February 25, 1956, commencing at 1.30 p.m. The following contributions will be presented: "Cutaneous Manifestation of Internal Disease", Dr. I. O. Stahle; "Pyrexia in Childhood", Dr. V. L. Collins; "The Differential Diagnosis of Cardiac Pain", Dr. L. E. Hurley; "The Management of Apoplexy",

Dr. J. B. Curtis; "Obesity", Dr H. P. Taft; "The Problem of the Refractory Anæmia", Dr. C. de Gruchy.

Dr. George Strong, President of the American College of Physicians, will deliver a lecture on "Rehabilitation: Experience in a New Rehabilitation Centre in Vancouver" in the lecture theatre at the Royal Melbourne Hospital at 8.30 p.m. on Wednesday, March 14, 1956.

All members of the British Medical Association are invited to attend both the scientific meeting and the lecture.

# Medical Practice.

## NATIONAL HEALTH ACT.

THE following notices appeared in the *Commonwealth of Australia Gazette*, Number 2, of January 12, 1956.

NATIONAL HEALTH ACT, 1953-1955.

**Part IV: Pensioner Medical Service.**

***Reprimand of Medical Practitioner.***

I, EARLE PAGE, the Minister of State for Health, hereby give notice in pursuance of sub-section (1.) of section 134A of the *National Health Act*, 1953-1955, that I have this day reprimanded Kenneth Boulton Shallard of Acacia-street, Killarney, medical practitioner, following investigation and report by the Medical Services Committee of Inquiry for the State of Queensland established under section 110 of the *National Health Act*, 1953, concerning the conduct of the aforesaid medical practitioner in relation to his provision of medical services for pensioners and their dependants under Part IV. of the *National Health Act*, 1953-1955, or under an arrangement made by the Director-General of Health under section 7 of the *National Health Service Act*, 1948-1949, and the National Health (Medical Services to Pensioners) Regulations.

Dated this twelfth day of December, 1955.

EARLE PAGE,
Minister of State for Health.

I, EARLE PAGE, the Minister of State for Health, hereby give notice in pursuance of sub-section (1.) of section 134A, of the *National Health Act*, 1953-1955, that I have this day reprimanded Kevin Hector Courtenay of 52 Oxford-street, Woollahra, medical practitioner, following investigation and report by the Medical Services Committee of Inquiry for the State of New South Wales established under section 110 of the *National Health Act*, 1953, concerning the conduct of the aforesaid medical practitioner in relation to his provision of medical services for pensioners and their dependants under Part IV. of the *National Health Act*, 1953-1955, or under an arrangement made by the Director-General of Health under section 7 of the *National Health Service Act*, 1948-1949, and the National Health (Medical Services to Pensioners) Regulations.

Dated this 16th day of December, 1955.

EARLE PAGE,
Minister of State for Health.

**Part VII: Pharmaceutical Benefits.**

***Suspension of Medical Practitioner.***

I, EARLE PAGE, the Minister of State for Health, hereby give notice in pursuance of sub-section (1.) of section 134A of the *National Health Act*, 1953-1955, that I have suspended until 5th June, 1956,

(*a*) the authority under section 88 of the said Act to write a prescription for the supply of pharmaceutical benefits;

(*b*) the approval under section 92 of the said Act; and

(*c*) the authority under section 93 of the said Act to supply pharmaceutical benefits

of Thomas H. Torr of Bute, South Australia, medical practitioner, following investigation and report by the Medical Services Committee of Inquiry for the State of South Australia established under the *National Health Act*, 1953-1955, concerning the conduct of the aforesaid medical practitioner in relation to his said authorities and approval and the provisions of the regulations under the said Act.

Dated this 20th day of December, 1955.

EARLE PAGE,
Minister of State for Health.

# Post-Graduate Work.

## THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

**Film Appraisal Group.**

APPLICATIONS are invited from those interested in joining the Film Appraisal Group of the Committee. Film appraisal evenings are held at regular times throughout the year, and the Committee would like to receive the names of those interested in attending.

## THE ROYAL INSTITUTE OF PUBLIC HEALTH AND HYGIENE.

**Medical Post-Graduate Courses of Instruction for Doctors.**

THE Royal Institute of Public Health and Hygiene conducts a recognized course of instruction (for post-graduate medical men and women only) for the Certificate in Public Health examination of the Conjoint Board of the Royal College of Physicians of London and the Royal College of Surgeons of England. This leads to courses for the Diploma in Public Health and for the Diploma in Industrial Health. Students are also prepared for the Diploma in Industrial Health examination of the Society of Apothecaries of London.

The next course of instruction for the Certificate in Public Health will commence on March 16, 1956. Further information, entry forms and prospectuses may be obtained from the Secretary of the Institute, 28 Portland Place, London, W.1, or from the Acting Dean at 23 Queen Square, London, W.C.1.

# Australian Medical Board Proceedings.

## NEW SOUTH WALES.

THE following additions and amendments have been made to the Register of Medical Practitioners for New South Wales in accordance with the *Medical Practitioners Act*, 1938-1955:

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (*a*) of the Act: Melville, Hugh James, M.B., B.S., 1951 (Univ. Melbourne); McMiken, John Bell, M.B., Ch.B., 1925, M.D., 1942 (Univ. New Zealand), M.R.A.C.P., 1938, D.D.R., 1941 (Univ. Melbourne), D.T.R.E., 1941 (Univ. Melbourne).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (*b*) of the Act: Billington, Michael David, M.R.C.S. (England), 1951, L.R.C.P. (London), 1951; Carew-Smyth, Raymond Ponsonby, L.R.C.P. and S. (Edinburgh), 1930, L.R.F.P.S. (Glasgow), 1930; Carroll, James Thomas, M.B., B.Ch. (N.U. Ireland), 1941, F.R.C.S. (Ireland), 1952, D.R.C.O.G. (London), 1943; Hammond, Christine Elizabeth Elsie, M.B., B.S., 1952 (Univ. London), D.C.H. (England), 1955; MacLachlan, Ian, M.B., Ch.B., 1953 (Univ. Edinburgh); O'Brien, William Aloysius, M.B., B.Ch., 1952 (N.U. Ireland).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (*c*) of the Act: Cranston, Ernst; Kreuzburg, Lydia; Krivanek, Joseph; Reni, Henry.

Registered medical practitioner who is required to complete twelve months' hospital service in accordance with the provisions of Section 17 (3) and is registered under Section 17 (1) (*b*) of the Act: Grima, Carmel Paul, M.D., 1955 (Malta).

Registered medical practitioners who are required to complete twelve months' hospital service in accordance with the provisions of Section 17 (3) and are registered under Section 17 (1) (*c*) of the Act: Brandson, Andrew Samuel; Szuchewycz, Iwan.

The following additional qualifications have been registered: Abbott, Lewsbe George, D.D.M., 1955 (Univ. Sydney); Bell, David Samuel, B.Sc. (Med.), 1955 (Univ. Sydney); Bell, James, M.C.R.A., 1955; Holt, Raymond Stuart, B.Sc. (Med.),

## DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 31, 1955.[1]

| Disease. | New South Wales. | Victoria. | Queensland. | South Australia. | Western Australia. | Tasmania.[2] | Northern Territory. | Australian Capital Territory. | Australia.[3] |
|---|---|---|---|---|---|---|---|---|---|
| Acute Rheumatism | .. | .. | 3(2) | .. | .. | .. | .. | .. | 3 |
| Amœbiasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Ancylostomiasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Anthrax | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Bilharziasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Brucellosis | .. | .. | 1 | .. | .. | .. | .. | .. | 1 |
| Cholera | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Chorea (St. Vitus) | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Dengue | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Diarrhœa (Infantile) | 1 | 4(3) | 1(1) | .. | .. | .. | .. | 2 | 8 |
| Diphtheria | 1 | 1(1) | .. | .. | 3(3) | .. | .. | .. | 5 |
| Dysentery (Bacillary) | .. | .. | 4(4) | .. | 1 | .. | .. | .. | 5 |
| Encephalitis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Filariasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Homologous Serum Jaundice | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Hydatid | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Infective Hepatitis | 39(21) | 45(26) | .. | .. | 3(2) | .. | 1 | .. | 88 |
| Lead Poisoning | .. | .. | 1 | .. | .. | .. | .. | .. | 1 |
| Leprosy | .. | .. | 1 | .. | .. | .. | .. | .. | 1 |
| Leptospirosis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Malaria | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Meningococcal Infection | 1 | .. | 1 | .. | .. | .. | .. | .. | 2 |
| Ophthalmia | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Ornithosis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Paratyphoid | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Plague | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Poliomyelitis | 2 | .. | .. | .. | 8(4) | .. | .. | .. | 10 |
| Puerperal Fever | 1 | .. | 1(1) | .. | 1 | .. | .. | .. | 3 |
| Rubella | .. | 114(30) | .. | .. | .. | .. | .. | .. | 114 |
| Salmonella Infection | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Scarlet Fever | 7(7) | 4(2) | 6(3) | .. | 1 | .. | .. | .. | 18 |
| Smallpox | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Tetanus | .. | .. | 1(1) | .. | .. | .. | .. | .. | 1 |
| Trachoma | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Trichinosis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Tuberculosis | 43(25) | 6(5) | 1 | .. | 1 | .. | 2 | .. | 53 |
| Typhoid Fever | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Typhus (Flea-, Mite- and Tick-borne) | .. | .. | .. | .. | 1 | .. | .. | .. | 1 |
| Typhus )Louse-borne) | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Yellow Fever | .. | .. | .. | .. | .. | .. | .. | .. | .. |

[1] Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Tasmania.

## DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JANUARY 7, 1956.[1]

| Disease. | New South Wales. | Victoria. | Queensland. | South Australia. | Western Australia. | Tasmania. | Northern Territory. | Australian Capital Territory. | Australia. |
|---|---|---|---|---|---|---|---|---|---|
| Acute Rheumatism | 4(3) | 1(1) | 3(1) | .. | 1(1) | .. | .. | .. | 9 |
| Amœbiasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Ancylostomiasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Anthrax | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Bilharziasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Brucellosis | .. | 1(1) | .. | .. | .. | .. | .. | .. | 1 |
| Cholera | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Chorea (St. Vitus) | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Dengue | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Diarrhœa (Infantile) | 2(1) | 17(15) | 6(5) | .. | .. | .. | .. | .. | 25 |
| Diphtheria | 2(2) | 3(2) | 1 | .. | 5(2) | .. | .. | .. | 11 |
| Dysentery (Bacillary) | .. | .. | 5(4) | .. | 5(1) | .. | .. | .. | 10 |
| Encephalitis | .. | 1 | .. | 3(3) | .. | .. | .. | .. | 4 |
| Filariasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Homologous Serum Jaundice | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Hydatid | .. | 1 | .. | .. | .. | .. | .. | .. | 1 |
| Infective Hepatitis | 62(32) | 87(50) | .. | 9(4) | 2(2) | .. | 4 | .. | 164 |
| Lead Poisoning | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Leprosy | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Leptospirosis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Malaria | .. | .. | .. | .. | 1(1) | .. | .. | .. | 1 |
| Meningococcal Infection | .. | 2(2) | .. | 1(1) | 1(1) | .. | .. | .. | 4 |
| Ophthalmia | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Ornithosis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Paratyphoid | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Plague | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Poliomyelitis | 3(2) | 7(6) | 2(2) | 5(1) | 7(5) | .. | .. | .. | 24 |
| Puerperal Fever | .. | .. | .. | .. | .. | .. | 1 | .. | 1 |
| Rubella | .. | 134(110) | .. | 10(9) | .. | .. | .. | .. | 144 |
| Salmonella Infection | .. | .. | .. | 2(2) | .. | .. | .. | .. | 2 |
| Scarlet Fever | 9(9) | 18(12) | 3(1) | 1(1) | .. | .. | .. | .. | 31 |
| Smallpox | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Tetanus | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Trachoma | .. | .. | .. | .. | 1 | .. | .. | .. | 1 |
| Trichinosis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Tuberculosis | 28(18) | 21(17) | 5(3) | 3(3) | 2(2) | 6(5) | .. | .. | 65 |
| Typhoid Fever | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Typhus (Flea-, Mite- and Tick-borne) | 2 | .. | 1 | .. | .. | .. | .. | .. | 3 |
| Typhus (Louse-borne) | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Yellow Fever | .. | .. | .. | .. | .. | .. | .. | .. | .. |

[1] Figures in parentheses are those for the metropolitan area.

1954 (Univ. Sydney); Johnston, George Arthur William, M.R.A.C.P., 1940; Lynch, Gilbert Edward, F.R.C.S. (England), 1954; Myers, William Keith, F.R.C.P. (Edinburgh), 1955; Peipman, Eskil Vaino, M.S., 1950 (Univ. Sydney), F.R.C.S. (England), 1955; Pook, William, M.C.R.A., 1949; King, Raymond Henry, D.D.M., 1955; Lehmann, Geoffrey Maxwell, D.P.M., 1955 (Univ. Sydney).

## Notice.

### REGISTER OF TESTING LABORATORIES.

THE "Register of Laboratories" published by the National Association of Testing Authorities provides up-to-date information on the tests performed in almost two hundred laboratories throughout the Commonwealth. It shows complete details of tests performed in each laboratory registered by the Association and the availability of laboratory facilities to the community. It is a valuable source of information for those interested in physical or chemical testing of raw materials or products, precision measurement or calibration of instruments and equipment. Copies of the Register may be obtained from the National Association of Testing Authorities, 58 Margaret Street, Sydney, at a cost of £1. The information provided by the Register is kept current by means of amendment sheets, which are circulated free of charge by the Association at frequent intervals.

## Deaths.

THE following death has been announced:

GOTT.—Henry Gott, on January 9, 1956, at Sydney.

## Nominations and Elections.

THE undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Caust, Leslie James, M.B., B.S., 1953 (Univ. Adelaide), 47 Hopetoun Road, Toorak, Victoria.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Allen, Bruce Mitchell (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Ashley, Brewster Charles Earnfaunce (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Barry, Grahame Maxwell (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Basser, Leon Samuel, M.B., B.S., 1946 (Univ. Sydney); Cookson, Philippa Nancy (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Coulthurst, Keith Dudley, M.B., B.S., 1955 (Univ. Sydney); Fry, Hunter John Hall (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Howe, Geoffrey David (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Hughes, Peter Robert (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Jackson, Jacqueline Ann (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Lewis, John Blake Paviour, M.B., B.S., 1955 (Univ. Sydney); Loudon, Richard Derby Kingsford, M.B., B.S., 1955 (Univ. Sydney); Marks, Harry (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Nott, David Bruce (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Oakeshott, Robert John (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Owen, Michael Samuel (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Oystragh, Philip (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Pittar, Yorke Arthur (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Pryde, Ian Noel (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Purchas, James Maurice (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Reading, Bruce David (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Standish, Patricia Anne (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Tottenham, Ronald Charles (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Walshe, Isobel Margaret Ann (provisionally registered), M.B., B.S., 1956 (Univ. Sydney); Toch, Walter Vojtech, regional registration for practice in the Urbenville-Woodenbong Region.

## Honours.

THE following promotions and admissions have been announced in the Venerable Order of the Hospital of Saint John of Jerusalem:

Dr. G. G. L. Stening and Dr. Morris Humphery, of New South Wales, have been promoted to the grade of Knight of Grace.

Dr. Robert S. Steel, O.B.E., of New South Wales, and Dr. A. L. Dawkins, O.B.E., Dr. D. D. McCowan and Dr. H. W. Ward, of Western Australia, have been promoted to the grade of Commander Brother.

Dr. W. E. George, of New South Wales, and Dr. I. O. Thorburn, of Western Australia, have been promoted to the grade of Officer Brother.

Dr. Robert J. Walsh, Dr. Eric H. Freidman and Dr. Frederick Tooth, of New South Wales, have been admitted in the grade of Serving Brother.

## Diary for the Month.

FEB. 1.—Victorian Branch, B.M.A.: Branch Meeting.
FEB. 3.—Queensland Branch, B.M.A.: General Meeting.
FEB. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee, 8 p.m. (with Special Groups 8.30 p.m.).
FEB. 10.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 10.—Tasmanian Branch, B.M.A.: Council Meeting.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

*New South Wales Branch* (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

*Queensland Branch* (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

*South Australian Branch* (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

*Western Australian Branch* (Honorary Secretary, 8 King's Park, West Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

## Editorial Notices.

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